





1899-1900



M616.06  
N48

PRESENTED BY  
RICHARD MILLS PEARCE  
AND BY  
THE JOHN HERR MUSSER  
DEPARTMENT OF  
RESEARCH MEDICINE

DEPARTMENT OF  
RESEARCH IN MEDICINE  
UNIVERSITY OF PENNSYLVANIA  
PHILADELPHIA, PA.

CANCELLED

6





S

DEPARTMENT OF  
RESEARCH MEDICINE  
UNIVERSITY OF PENNSYLVANIA  
PHILADELPHIA, PA.  
**CANCELLED**





*A  
Mell  
no*

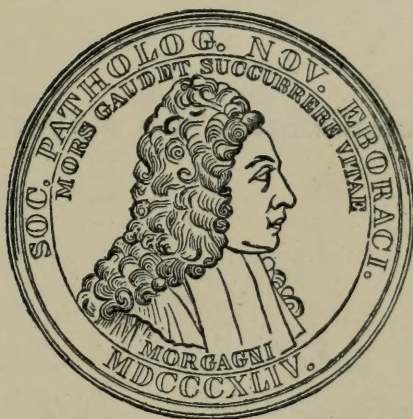
# PROCEEDINGS

OF THE

NEW YORK

## PATHOLOGICAL SOCIETY

FOR THE YEARS 1899 AND 1900



ORGANIZED IN 1844

INCORPORATED IN 1886

PRINTED FOR THE SOCIETY

1901

437794  
1.8.45

UNIVERSITY  
OF PENNSYLVANIA  
LIBRARY

DEPARTMENT OF  
RESEARCH IN MEDICINE  
UNIVERSITY OF PENNSYLVANIA  
PHILADELPHIA, PA.  
~~CANCELLED~~

RB

1

N32

1899-

1900

The Knickerbocker Press, New York

UNIVERSITY  
OF  
PENNSYLVANIA  
LIBRARY



## CONTENTS.

	PAGE
ILLUSTRATIONS . . . . .	v
LIST OF OFFICERS AND COMMITTEES FOR THE YEAR 1899 .	vii
LIST OF OFFICERS AND COMMITTEES FOR THE YEAR 1900 .	viii
PRESIDENTS OF THE SOCIETY . . . . .	ix
SECRETARIES OF THE SOCIETY . . . . .	x
LIFE MEMBERS OF THE SOCIETY . . . . .	xi
MEMBERS OF THE SOCIETY . . . . .	xiii
MEMBERS DECEASED IN 1899 AND 1900 . . . . .	xix
PROCEEDINGS OF THE SOCIETY . . . . .	i
MIDDLETON-GOLDSMITH LECTURE: "THE ETIOLOGY OF TROPICAL DYSENTERY," BY DR. SIMON FLEXNER .	297
INDEX. . . . .	339





## ILLUSTRATIONS

---

	PAGE
MILIARY TUBERCLES OF THE PLEURA . . . . .	46
MILIARY TUBERCLES OF THE PLEURA . . . . .	47
PLEURA STUDDED WITH MILIARY TUBERCLES. A TU- BERCULOUS PATCH NEAR THE APEX . . . . .	47
A MILIARY AIR CYST OF THE PLEURA . . . . .	49
A CONGLOMERATE TUBERCLE OF THE PLEURA . . . . .	49
A MILIARY TUBERCLE OF THE PLEURA, SURROUNDED WITH COAL-DUST PIGMENT . . . . .	49
AN ENCAPSULATED MILIARY TUBERCLE OF THE PLEURA	50
A MILIARY TUBERCLE OF THE PLEURA, CONTAINING A GIANT CELL . . . . .	51
A CHEESY TUBERCLE OF THE PLEURA . . . . .	51
DIFFUSE TUBERCLE TISSUE OF THE PLEURA . . . . .	51





# OFFICERS AND COMMITTEES FOR THE YEAR 1899.

---

*President,*

DR. T. MITCHELL PRUDDEN.

*Vice-President,*

DR. W. H. PARK.

*Treasurer,*

DR. JOHN H. HINTON.

*Secretary,*

DR. OGDEN C. LUDLOW.

*Editor,*

DR. ROWLAND G. FREEMAN.

*Trustees,*

DR. JOHN H. HINTON,

DR. T. MITCHELL PRUDDEN,

DR. EDWARD K. DUNHAM,

DR. GEORGE C. FREEBORN,

DR. HERMAN M. BIGGS,

DR. GEORGE P. BIGGS.

---

## COMMITTEE ON ADMISSIONS AND ETHICS.

DR. GEORGE P. BIGGS,

DR. VAN HORNE NORRIE,

DR. JOHN H. LARKIN,

DR. JAMES EWING,

DR. HARLOW BROOKS.

---

## COMMITTEE ON PUBLICATION.

DR. E. K. DUNHAM,

DR. D. H. McALPIN, JR.,

DR. ROWLAND G. FREEMAN, *Editor,*

DR. JOHN H. HINTON, *Treasurer,*

DR. OGDEN C. LUDLOW, *Secretary.*

---

## COMMITTEE ON MICROSCOPY.

DR. G. A. TUTTLE,

DR. HARLOW BROOKS,

DR. LEON T. LEWALD.

# OFFICERS AND COMMITTEES FOR THE YEAR 1900.

---

## *President,*

DR. EUGENE HODENPYL.

## *Vice-President,*

DR. JAMES EWING.

## *Treasurer,*

DR. JOHN H. HINTON.

## *Secretary,*

DR. OGDEN C. LUDLOW.

## *Editor,*

DR. ROWLAND G. FREEMAN.

## *Trustees,*

DR. GEORGE C. FREEBORN,

DR. GEORGE P. BIGGS,

DR. E. K. DUNHAM,

DR. T. MITCHELL PRUDDEN,

DR. JOHN H. HINTON,

DR. D. H. McALPIN, JR.

---

## COMMITTEE ON ADMISSIONS AND ETHICS.

DR. GEORGE P. BIGGS,

DR. JAMES EWING,

DR. VAN HORNE NORRIE,

DR. JOHN H. LARKIN,

DR. HARLOW BROOKS.

---

## COMMITTEE ON PUBLICATION.

DR. E. K. DUNHAM,

DR. D. H. McALPIN, JR.,

DR. ROWLAND G. FREEMAN, *Editor,*

DR. JOHN H. HINTON, *Treasurer,*

DR. OGDEN C. LUDLOW, *Secretary.*

---

## COMMITTEE ON MICROSCOPY.

DR. EDWARD K. DUNHAM,

DR. JAMES EWING,

DR. J. S. THACHER.



## PRESIDENTS OF THE SOCIETY.

---

* DR. JOHN A. SWETT,	1844
* DR. WILLARD PARKER,	1845, 1846, 1847
* DR. JAMES R. WOOD,	1848, 1857
DR. T. M. MARKOE,	1850, 1879
* DR. W. H. VAN BUREN,	1850
* DR. CHARLES E. ISAACS,	1851
DR. JOHN T. METCALFE,	1852
* DR. HENRY VAN ARSDALE,	1853
* DR. JACKSON BOLTON,	1854, 1855
* DR. ROBERT WATTS,	1856
* DR. EDMUND R. PEASLEE,	1858
* DR. JOHN C. DALTON,	1859
* DR. ALFRED C. POST,	1861
* DR. THOMAS C. FINNELL,	1862
* DR. DAVID S. CONANT,	1863
DR. ABRAHAM JACOBI,	1864
* DR. GURDON BUCK,	1865
* DR. HENRY B. SANDS,	1866
* DR. WILLIAM B. BIBBINS,	1867
* DR. ERNEST K. KRACKOWITZER,	1868
DR. L. A. SAYRE,	1869
* DR. JOSEPH C. HUTCHINSON,	1870
* DR. A. L. LOOMIS,	1871, 1872
* DR. ERSKINE MASON,	1873
DR. HERMAN KNAPP,	1874
DR. FRANCIS DELAFIELD,	1875
DR. CHARLES K. BRIDDON,	1876
DR. EDWARD G. JANEWAY,	1877
* DR. JOHN C. PETERS,	1878
DR. EDWARD L. KEYES,	1879
DR. T. E. SATTERTHWAITE,	1880, 1881

\* Deceased.

* DR. E. C. SEGUIN,	1882
DR. GEORGE F. SHRADY,	1883, 1884
DR. JOHN A. WYETH,	1885, 1886
DR. T. MITCHELL PRUDDEN,	1887, 1898, 1899
DR. W. P. NORTHRUP,	1888, 1889
* DR. J. WEST ROOSEVELT,	1890
DR. HERMAN M. BIGGS,	1891
DR. H. P. LOOMIS,	1892, 1893
DR. G. C. FREEBORN,	1894
DR. GEORGE P. BIGGS,	1895
DR. JOHN SLADE ELY,	1896, 1897
DR. EUGENE HODENPYL,	1900

---

## SECRETARIES.

---

* DR. WILLIAM C. ROBERTS,	1844 to 1849
* DR. H. D. BUCKLEY,	1849
* DR. HENRY G. COX,	1850 to 1852
* DR. WILLIAM HENRY CHURCH,	1852
* DR. CHARLES M. ALLEN,	1852 to 1853
* DR. GEORGE T. ELLIOTT,	1853 to 1854
* DR. J. FOSTER JENKINS,	1854 to 1855
* DR. E. LEE JONES,	1855 to 1861
DR. T. GAILLARD THOMAS, <i>pro tem.</i> ,	1855
* DR. HENRY D. NOYES, <i>pro tem.</i> ,	1858
DR. GEORGE F. SHRADY,	1861 to 1879
* DR. WESLEY M. CARPENTER,	1880 to 1888
DR. WALTER MENDELSON,	1889
DR. T. L. STEDMAN,	1889 to 1891
DR. OGDEN C. LUDLOW,	1891 to 1900

\* Deceased.

## LIFE MEMBERS.

---

Elected.	Address.
1861 BLUMENTHAL, MARK .....	Europe
1865 BRADLEY, EDWARD.....19	West 30th Street
1867 DELAFIELD, FRANCIS.....12	West 32d Street
1858 DRAPER, WILLIAM H.....19	East 47th Street
1855 ELIOT, ELLSWORTH.....48	West 36th Street
1853 EMMET, THOMAS ADDIS.....91	Madison Avenue
1856 HINTON, JOHN H.....41	West 32d Street
1862 HULL, JOSEPH J.....47	West 78th Street
1861 JACOBI, ABRAHAM.....110	West 34th Street
1867 JANEWAY, EDWARD G.....36	West 40th Street
1868 KNAPP, HERMAN.....26	West 40th Street
1858 LAMBERT, EDWARD W.....2	East 37th Street
1869 LEALE, C. A.....604	Madison Avenue
1847 MARKOE, THOMAS MASTERS.....500	Madison Avenue
1869 NEFTTEL, W. B.....16	East 48th Street
1864 NEWMAN, ROBERT.....148	West 73d Street
1858 NICOLL, H. D.....51	East 57th Street
1858 PACKARD, CHARLES W.....447	Park Avenue
1866 PURDY, ALFRED E. M.....304	Madison Avenue
1858 SHRADY, GEORGE F.....8	East 66th Street
1855 THOMAS, THEODORE GAILLARD...600	Madison Avenue
1857 WARNER, J. W.....	Saratoga Springs, N. Y.
1868 WYNKOOP, GERARDUS H.....128	Madison Avenue





## MEMBERS OF THE SOCIETY.

Elected.	Address.
1877 Abbé, Robert.....	13 West 50th Street
1890 Adams, Calvin Thayer.....	21 East 28th Street
1894 Adams, Robert Staunton.....	12 West 33d Street
1898 Adler, Isaac.....	12 East 60th Street
1891 Alexander, Samuel.....	5 West 58th Street
1898 Ashly, Dexter D.....	256 West 91st Street
1900 Assenheimer, Augustus.....	323 East 51st Street
1897 Auzal, Ernest W.....	23 West 53d Street
1898 Bailey, F. R.....	437 West 59th Street
1893 Bailey, Pearce.....	4 West 50th Street
1881 Bang, Richard T.....	139 West 11th Street
1895 Barstow, Donald M.....	21 East 53d Street
1900 Bates, W. H.....	50 East 64th Street
1892 Beach, Bennett S.....	210 West 34th Street
1896 Belcher, Sarah.....	542 Fifth Avenue
1900 Berkeley, W. N.....	121 East 26th Street
1891 Biggs, George Patten.....	39 East 63d Street
1887 Biggs, Herman M.....	5 West 58th Street
1901 Bishop, Louis Fougères.....	54 West 55th Street
1886 Bissell, Joseph B.....	15 West 58th Street
1889 Bleything, George D.....	66 East 77th Street
1885 Boldt, Hermann J.....	54 West 51st Street
1896 Bovaird, David.....	126 West 58th Street
1888 Brannan, John Winters.....	11 West 12th Street
1896 Brickner, Samuel M.....	136 West 85th Street
1901 Brill, N. E.....	125 West 77th Street
1896 Brooks, Harlow.....	44 West 9th Street
1899 Brown, Alice Crawford. 80 State St., Hackensack, N. J.	
1888 Brown, F. Tilden.....	141 East 58th Street
1880 Cammann, D. M.....	19 East 33d Street

1887	Carr, Walter Lester.....	68 West 51st Street
1889	Carter, DeLancy.....	1030 Park Avenue
1900	Carter, Herbert S.....	36 West 55th Street
1879	Chambers, Potter Flewellen.....	49 East 57th Street
1887	Cheesman, Timothy Matlack....	46 East 29th Street
1891	Coleman, Warren.....	5 West 30th Street
1893	Coley, William B.....	5 Park Avenue
1895	Conner, Lewis A.....	48 West 49th Street
1898	Cordes, Louise.....	46 East 49th Street
1891	Currier, Charles Gilman.....	313 West 102d Street
1877	Cushier, Elizabeth M.....	53 East 20th Street
1880	Delavan, D. Bryson.....	1 East 33d Street
1896	Denton, Myron Preston.....	33 East 33d Street
1884	Dixon, George A.....	15 West 49th Street
1900	Dow, E. L.....	49 West 57th Street
1889	Dowd, Charles N.....	135 West 73d Street
1889	Dunham, Edward K.....	338 East 26th Street
1880	Edebohls, George M.....	59 West 49th Street
1888	Elliott, George T.....	36 East 35th Street
1900	Evans, Evan M.....	2 West 83d Street
1893	Ewing, James.....	260 West 57th Street
1882	Ferguson, Frank.....	20 West 38th Street
1895	Fischer, Charles S.....	152 West 57th Street
1889	Fisher, Edward D.....	42 West 45th Street
1895	Fitzpatrick, Charles B., Rosebank, Bor. of Richmond, N. Y. City.	
1892	Fordyce, John A.....	66 Park Avenue
1885	Freeborn, George C.....	215 West 70th Street
1888	Freeman, Rowland Godfrey....	205 West 57th Street
1892	Fuller, R. M.....	136 West 42d Street
1874	Gibney, Virgil P.....	16 Park Avenue
1889	Grauer, Frank.....	333 West 46th Street
1887	Hamilton, C. S., 142 East Long Street, Columbus, O.	
1899	Hart, J. Stuart.....	77 West 50th Street



1899	Hart, Theo. S.....	130	West 59th Street
1896	Heiman, Henry.....	56	West 120th Street
1878	Heineman, Henry N.....	62	West 51st Street
1884	Henry, Nelson H.....	48	East 11th Street
1889	Herter, Christian A.....	819	Madison Avenue
1900	Herzfeld, Alfred A.....	214	West 24th Street
1900	Hiss, Philip Hanson.....	28	Washington Square, N.
1886	Hodenpyl, Eugene.....	143	West 73d Street
1897	Holder, Oscar H.....	66	Park Avenue
1882	Holt, L. Emmett.....	14	West 55th Street
1900	Hubbard, E. V.....	138	West 74th Street
1893	Huddleston, John H.....	126	West 85th Street
1862	Hull, J. J.....	51st St., and Broadway	
1880	Jackson, Frank W.....	12	West 18th Street
1872	Jacobi, Mary Putnam.....	110	West 34th Street
1896	James, Robert C.....	5	West 30th Street
1887	James, Walter B.....	17	West 54th Street
1898	Janeway, Theodore C.....	36	West 40th Street
1894	Jeffries, Ferdinand M.....	821	East 165th Street
1900	Jelliffe, Smith Ely.....	231	West 71st Street
1900	Jessup, Stuart D.....	356	West 21st Street
1887	Jones, Mary A. D.....	249	East 86th Street
1879	Judson, Adoniram B.....	1	Madison Avenue
1887	Kilham, Eleanor B.....	121	East 35th Street
1875	Kipp, Charles J....	534	Broad Street, Newark, N. J.
1898	Knapp, Arnold.....	26	West 50th Street
1891	Kneer, Ferdinand G.....	236	West 51st Street
1884	Knight, Charles H.....	147	West 57th Street
1889	Koerner, C. F.....	242	East 12th Street
1893	Lambert, Alexander.....	125	East 36th Street
1879	Lange, Fred.....	130	East 61st Street
1899	Langmann, Gustav.....	121	West 57th Street
1896	Larkin, John Henry.....	498	West 130th Street
1899	Lartigau, August Jerome.....	231	West 71st Street
1896	Leaming, Edward.....	117	West 84th Street

1883	LeBoutillier, William G.....	49	West 50th Street
1891	LeFevre, Egbert.....	52	West 56th Street
1898	Levene, Phœbus A.....	1692	Lexington Avenue
1898	Levin, Isaac.....	1696	Lexington Avenue
1898	LeWald, Leon T.....	55	West 36th Street
1898	Lewis, Charles H.....	47	West 58th Street
1880	Lewis, Daniel.....	252	Madison Avenue
1899	Libman, Emanuel.....	180	East 64th Street
1885	Lockwood, George Roe, Jr.....	44	West 49th Street
1899	Long, Eli.....	69	West 97th Street
1885	Loomis, H. P.....	58	West 34th Street
1890	Ludlow, Ogden Curtis.....	2309	Seventh Avenue
1889	Lynch, John B.....	148	West 22d Street
1888	Lynde, George S.....	326	West 45th Street
1889	MacHale, Ferdinand S.....	317	East 87th Street
1891	McAlpin, D. Hunter, Jr.....	9	East 55th Street
1900	McLaughlin, George E.....	41	Crescent Avenue, Jersey City, N. J.
1878	McNutt, Sarah J.....	265	Lexington Avenue
1895	Mandelbaum, F. S.....	717	Madison Avenue
1900	Mannheimer, George.....	183	East 80th Street
1884	Markoe, Frances H.....	15	East 49th Street
1889	Markoe, J. W.....	12	West 55th Street
1898	Mathews, F. S.....	62	West 58th Street
1878	Mayer, Abraham.....	40	East 60th Street
1897	Mercelis, Elizabeth.....	65	Church St., Montclair, N. J.
1886	Meyer, Willy.....	700	Madison Avenue
1874	Moeller, Henry.....	341	West 57th Street
1898	Moschcowitz, Alexis V.....	350	West 58th Street
1890	Mowry, Eugene C.....	355	West 42d Street
1887	Myers, T. Halsted.....	54	West 50th Street
1892	Myles, Robert C.....	46	West 38th Street
1864	Newman, R.....	148	West 73d Street
1896	Nicoll, Matthias, Jr.....	168	West 48th Street
1894	Norrie, Van Horne.....	21	West 37th Street
1896	Norris, Charles.....	23	East 39th Street

1883	Northrup, William P.....	57 East 79th Street
1895	Noyes, William B.....	28 West 61st Street
1899	Oertel, Horst.....	26th St. and First Ave.
1893	Park, William Hallock.....	315 West 76th Street
1876	Partridge, Edward L.....	19 Fifth Avenue
1886	Peterson, Frederick.....	4 West 50th Street
1898	Phillips, Carlin.....	39 West 27th Street
1874	Polk, William M.....	7 East 36th Street
1878	Porter, William H.....	1674 Broadway
1900	Potter, N. B.....	9 West 35th Street
1893	Power, Henry.....	Montclair, N. J.
1883	Prudden, T. Mitchell.....	437 West 59th Street
1887	Pryor, William Rice.....	121 East 38th Street
1884	Rice, Clarence C.....	123 East 19th Street
1881	Robinson, A. R.....	248 West 42d Street
1900	Sabine, Philip S.....	960 Madison Avenue
1891	Sachs, B.....	21 East 65th Street
1874	Satterlee, F. LeRoy.....	8 West 18th Street
1872	Satterthwaite, Thomas E.....	7 East 80th Street
1887	Sayre, Reginald H.....	285 Fifth Avenue
1900	Schlapp, M. G.....	122 East 62d Street
1897	Schultze, Otto H.....	1109 Madison Avenue
1890	Seaman, Louis L.....	18 West 31st Street
1887	Sellew, Frederick S.....	61 East 79th Street
1895	Shelby, Edmond P., Jr.....	30 West 32d Street
1899	Smith, Ernest Ellsworth.....	262 Fifth Avenue
1894	Sondern, Frederic E.....	200 West 56th Street
1889	Spitzka, E. C.....	66 East 73d Street
1888	Stedman, Thomas L.....	1425 Broadway
1898	Stein, Richard.....	811 Lexington Avenue
1872	Stimson, Daniel M.....	11 West 17th Street
1894	Stone, William S.....	1730 Broadway
1900	Stow, Bond.....	56 West 51st Street
1900	Summer, Albert E.....	122 West 58th Street
1880	Swasey, John H.....	34 East 28th Street



1886	Thacher, John Seymour.....	33	West 39th Street
1899	Thayer, A. E.....	352	West 117th Street
1896	Thelberg, John.....	26	West 34th Street
1896	Valadier, Charles A.....	130	East 64th Street
1888	Van Cott, J. M.....	188	Henry Street, Brooklyn
1886	Van Gieson, Ira.....	1	Madison Avenue
1882	Van Santvoord, Richard.....	106	West 122d Street
1885	Van Schaick, George Graf.....	23	West 37th Street
1883	Wackerhagen, G.....	23	Seventh Avenue, Brooklyn
1897	Wadsworth, Augustus.....	112	West 55th Street
1878	Walker, Henry Freeman.....	18	West 55th Street
1882	Walsh, Simon J.....	25	East 128th Street
1886	Warrin, M. L.....		France
1881	Wendt, Edmund Charles.....	118	West 79th Street
1882	Wiener, R. G.....	48	East 65th Street
1899	Wilcow, Ernest N.....	43	West 75th Street
1894	Williams, Anna W.....	352	West 117th Street
1891	Wollstein, Martha.....	321	East 15th Street
1898	Wood, Francis Carter.....	8	East 49th Street
1885	Wright, Jonathan.....	73	Remsen Street, Brooklyn
1881	Wylie, W. Gill.....	28	West 40th Street

During the Years 1899 and 1900  
the following Members were lost to  
the Society through Death

J. H. Linsley

John A. McCreery

Lewis A. Sayre

J. W. Stickler

J. C. Smith

Wm. Vissmann





PROCEEDINGS  
OF THE  
NEW YORK PATHOLOGICAL SOCIETY.

---

*Stated Meeting, February 8, 1899.*

T. MITCHELL PRUDDEN, M.D., PRESIDENT.

COMPLETE STENOSIS AND DILATATION OF THE APPENDIX.

Dr. A. V. MOSCHCOWITZ presented an appendix removed by operation three weeks previously, from a girl of twelve years, who had had, while under his observation, two attacks of appendicitis. One of these had occurred last July, and had not been characterized by very marked symptoms. At that time operation had been advised, but had not been strenuously urged. The second attack occurred in December, and had also been mild. Neither the temperature nor the pulse had been high, and the pain had been moderate. Recovery had taken place in a short time, and the operation had been done in the interval, and by the method of blunt division of the muscles. The appendix had been removed without difficulty. He had then been impressed with the remarkable thinness of the appendix at the attachment to the cæcum. He had made it a practice, in operating upon cases of appendicitis, to disinfect the

mucous membrane by the insertion of a fine point of the Paquelin cautery. This procedure had been impossible in the present instance, because the aperture would not admit even a fine probe. On cutting open the appendix a careful microscopic examination had shown the obliteration of its canal and disappearance of the mucous membrane and muscular layer, only the serous membrane and the connective tissue being left near its attachment to the cæcum, while the distal portion of the appendix was dilated in a balloon shape.

#### COMPLETE TRANSVERSE RUPTURE OF THE AORTA.

Dr. LEON T. LEWALD exhibited a specimen showing complete transverse rupture of the aorta. It had been taken from a woman, seventy years of age. The pericardium had been distended with blood, and there had been a rupture into the pericardial sac from the tissues around the root of the aorta, together with a complete transverse rupture of the middle and inner coats of the aorta, one inch above the aortic valves. The blood had dissected its way as far up as the bifurcation of the carotid arteries, and down into the heart itself behind the pericardium. Death had resulted from the pressure on the heart. There was no history of injury and no evidence of violence, so that the rupture had evidently been spontaneous. The patient had been found dead on the steps of a building, and it was supposed that she had died from exposure to cold, but as she had been suffering from bronchitis, it was possible that the rupture had occurred during an attack of coughing.

#### SPONTANEOUS RUPTURE OF THE INNER AND MIDDLE COATS OF THE AORTA WITH DISSECTING ANEURISM.

Dr. LEWALD also showed specimens from another case of rupture of the aorta. The subject was a male, fifty

years of age, who had died suddenly while eating. The autopsy had revealed a rupture through an atheromatous patch in the descending arch of the aorta. There had been extensive hemorrhage into the right pleural cavity, and a smaller rupture into the left pleural cavity. The blood had dissected between the outer and middle coats of the aorta and its large branches.

SPONTANEOUS RUPTURE OF THE TRANSVERSE ARCH OF  
THE AORTA WITH DISSECTING ANEURISM.

He also presented still another specimen of this condition, occurring in a woman about fifty years of age. The immediate cause of death had been rupture of the dissecting aneurism into the pericardial sac. The extravasation and dissecting aneurism in this case had also been very marked, particularly in the abdominal aorta. Dr. Theodore Janeway had looked up the literature of the subject, and had found eighteen similar cases, in all but two of which the rupture had been spontaneous. In thirteen out of the sixteen cases of spontaneous rupture, the rupture had taken place into the pericardium, in two into the mediastinum, and in one there had been no special extravasation. In four of the cases there had been complete rupture without dissecting aneurism, and one with a dissecting aneurism. Of the remaining eleven cases the rupture had been of the middle and inner coats, with the formation of dissecting aneurisms in all but one case. Death had been sudden in eleven of the sixteen cases mentioned. Fourteen of the cases had been males, and two females.

PULMONARY VALVE WITH FOUR CUSPS.

Dr. LEWALD then showed a pulmonary valve having three cusps of good size, and a fourth larger than the



others. Two of the valves were fenestrated. The specimen had been taken from a Chinaman, sixty years of age. No abnormal sound had been detected over the valve during life.

The PRESIDENT recalled a case formerly presented to the Society, in which, with a fresh rupture of the aorta and considerable extravasation, there was evidence of several earlier partial or complete ruptures of the vessel which had healed.

#### ADRENAL DEPOSITS IN THE LIVER.

Dr. W. B. NOYES presented gross and microscopic specimens of a liver which had been removed from an Italian, who had been treated in the Columbus Hospital for cirrhosis of the liver for a month before his death. The autopsy had revealed a hypostatic pneumonia, rather a typical, large, hobnail liver, and the lesions of chronic diffuse nephritis. The entire liver, besides presenting the ordinary appearance of a cirrhotic liver, contained a number of whitish nodules, about the size of an English walnut, and resembling areas of degeneration or possibly isolated gummata which had broken down. Under the microscope these nodules were found to be composed of glandular structure, bearing a very close resemblance to the suprarenal gland. In the periphery of the liver there was more normal tissue.

#### *Discussion.*

Dr. CARLIN PHILLIPS said that he had had an opportunity of examining this liver. On section, in the gross, it had presented a peculiar, mottled appearance, which at first did not suggest a secondary tumor formation. Scattered throughout the tissue were yellowish areas with indistinct outline, associated more especially with

the portal vein. Many of the veins themselves were filled with whitish material. These areas microscopically were made up of large clear cells with small round nuclei, which resembled in every way those cells from the zona fasciculata of the suprarenal body.

Dr. ISAAC ADLER said that he did not think this condition was quite as rare as would appear from consulting the text-books on pathology. Many specimens had doubtless been classed as adenomata when they had really been portions of the suprarenal gland. He had seen two undoubted cases of adrenal deposits in the liver. Such deposits in the pancreas had also been described.

A CASE OF ADDISON'S DISEASE, WITH SIMPLE ATROPHY OF  
THE ADRENAL BODY.

Dr. CARLIN PHILLIPS read a paper with this title, in which he reported the following case: A male, forty-two years of age, had been admitted to the City Hospital on August 10, 1897, with a negative family history. He had had his first attack of rheumatoid pains twenty years before, and stated that he had begun to turn yellow fourteen years ago. Six weeks before admission he had had an attack of weakness, associated with swelling of the feet and pains in the legs and ankles. At the time of entering the hospital his entire body had been of a yellowish color, which was especially dark over the areas pressed upon by his clothes. The ocular conjunctiva was pearly-white; the mucous membranes were very pale; he was mentally dull; the bowels moved three or four times a day. On September 11th he had had severe darting pains in the neck and occipital region, and more diarrhoea, followed by impaired resonance over both infraclavicular regions. On November 5, 1897, physical examination of the chest had revealed bron-



chial breathing and coarse mucous râles, but no cavity formation. Examination of the urine had been made at intervals, but with negative result. Death had occurred on February 17th. At the autopsy, which had been made a few hours after death, it was noted that there was extreme emaciation and that the skin of the entire body was of a brownish mulatto color, most marked on the anterior surface of the thorax, on the scrotum, head of the penis, and dorsum of the hands and feet. There was no pigment deposit on the buccal mucous membrane. The upper lobe of the right lung showed moderate anthracosis, while there were tuberculous nodules in the apex, and a few in the lower lobe of the left lung. The bronchial glands were enlarged and calcified. The pericardium contained about twenty cubic centimetres of fluid. The heart was of average size, and presented no valvular lesions, but the left ventricle was dilated and its walls were thin and of a pale brown color. The liver was small and reddish-brown; its lobules were easily seen and had darkish centres. The gall bladder was normal. The stomach was small, but otherwise normal. The pancreas was likewise small, and was the seat of interstitial hyperplasia. The mucous membrane of the small intestine was pale and covered with mucus. The mesenteric glands were small and firm. The spleen was somewhat enlarged. The right kidney was enlarged; its cortex was pale and yellowish, and its capsule stripped off, leaving a fine granular surface. The left suprarenal was two inches in length and three-fourths of an inch in width, by three-fourths of an inch in thickness. The right kidney was of the same dimensions, and appeared normal in every way. The surface was smooth and pale, and the consistency was normal. The medullary portion was of proper thickness, and there was no evidence of tuberculous disease. The pia



mater of the brain was extremely œdematous, opaque, and thickened. The brain itself was small, symmetrical, and very pale. On the under surface of the dura were small hemorrhagic extravasations. The left suprarenal was two inches in length, three-fourths of an inch in width, and one-fourth of an inch in thickness. The right suprarenal was of the same dimensions. The general appearance was normal in every way, possibly somewhat atrophic. The surface was smooth and pale; the consistency was normal. On cut section the medullary portion seemed to be of normal thickness relative to the cortex. No evidence of tuberculosis was present. The anatomical diagnosis was chronic miliary tuberculosis of the lungs and bronchial glands; subacute congestion of the liver; chronic interstitial pancreatitis; chronic catarrhal enteritis; chronic interstitial nephritis; simple atrophy of the adrenal bodies.

The microscopical study of the case had been conducted with the aid of a great variety of stainings. Many of the sections of the adrenal bodies appeared normal on casual examination, but more careful scrutiny showed a definite pathological state. The intracellular substance was homogeneous, finely granular, and in places fibrillated. The fibrous capsule was apparently not thickened, and was free from inflammation, tuberculous or otherwise. The individual glomeruli varied greatly in size and appearance. In places this zone of glomeruli appeared much thinner than normal; in others it was entirely absent. The portion of the gland represented by the zona fasciculata presented many changes; particularly the irregularity of the tubules, the branching columns of cells having an irregular course and being intimately mixed with the zona reticularis. The protoplasm was finely granular, and the walls of the individual cells could not be made out. The fatty changes so com-

monly seen in this region were noticeably diminished, there being very few of the large, clear, fatty cells so constantly seen in the normal organ. The most prominent departure from the normal was seen in the region adjacent to and including the upper medullary portion, or the zona reticularis. The cells here, instead of being in small anastomosing columns, remained more or less distinct, in groups of two or three — the most distinctive evidence of atrophy. The polymorphism of the cells was very noticeable. Many of the large homogeneous giant cells were seen. The protoplasm appeared nearly homogeneous and without definite limits. Many of the smaller cells of this zone also showed evidences of degeneration. The intracellular substance and fibrous reticulum were especially noticeable, probably because of the atrophy and disappearance of the cell columns in this portion. There was apparently no increase in the fibrous stroma of the gland. The reticulum throughout the gland was rich in nuclei, which stained deeply. The medulla of both glands showed but little evidence of disease. Here and there, as often seen in the normal organ, were portions of the cortex. At the junction of the medulla with the cortex were ganglion cells staining deeply with Nissl's stain. The right and left semilunar ganglia were stained in various ways. With Nissl's stain the cells appeared large and fully formed. The Nissl bodies were well formed, standing out clearly in the cytoplasm. Aside from the increase of the pigment there was no abnormality.

Summary of microscopical findings: (1) Diminution in size and number of glomeruli of the zona glomerulosa. (2) Diminution in length with great irregularity in course of the tubules in the zona fasciculata. (3) Diminution of the fatty content of the cells in the zona



fasciculata. (4) Colloid degeneration of circumscribed areas in the zona fasciculata. (5) Marked atrophy with increased pigmentation of cells in the zona reticularis. (6) General diminution in size of the medulla, with the presence of transposed cortical elements. (7) Presence of many mononuclear giant cells. (8) Thickening of the vessel walls with perivascular infiltration. (9) Complete absence of chronic interstitial or tuberculous inflammation.

Dr. PHILLIPS said that a review of the literature of Addison's disease seemed to indicate that, aside from tuberculous disease, simple atrophy was apparently the most common cause. Up to the present time, fourteen cases of simple atrophy of the suprarenal bodies had been reported, associated with symptoms of Addison's disease, the present case being the first reported in this country. Tuberculosis had been present three times, and in none of them had the changes in the glands been so insignificant as in the case just reported. In three of the cases the gland had been entirely absent. The pathogenesis of simple atrophy of the suprarenal gland was practically unknown. The case just reported was rather one of perversion than of total lack of function.

#### *Discussion.*

Dr. P. A. LEVENE said that the relation between disease of the suprarenal bodies and Addison's disease had not been fully established, and he understood that there were cases of Addison's disease on record without any pathological alteration of the suprarenal body. It was known that pigment could be produced by any proteid body, and hence such a formation could take place in any cell. He was disposed to believe that the chief symptom of Addison's disease—pigmentation—was a symptom of various diseases, just as glycosuria was only



a symptom of diabetes. The pigmentation might result from various causes, and this would explain the different findings in the suprarenal bodies.

Dr. ISAAC LEVIN said that, while it had been shown that most of the cases of Addison's disease had been accompanied by some alteration in the suprarenal bodies, in twelve per cent. they had been perfectly normal. On the other hand, there were cases in which the suprarenal bodies were the seat of cancer or other lesions, and yet these lesions were not accompanied by symptoms of Addison's disease. Physiology alone seemed competent to answer the question as to the relationship of the suprarenal bodies to Addison's disease. Physiology had demonstrated that extirpation of the adrenal gland caused death in fifteen hours, proving that the function of this gland was important to the organism. It has been further shown that neurin, a product of proteid metabolism found in the urine of patients with Addison's disease, is toxic if injected hypodermically into a frog. A substance allied to neurin had been found in the suprarenal bodies themselves. Thus this neurin may be the factor which will show us the true relation between Addison's disease and the suprarenal bodies.

A CASE OF WEIL'S DISEASE, WITH A SHORT EXPERIMENTAL  
STUDY OF INFECTIVE ICTERUS.

Dr. HARLOW BROOKS read this paper. He said that Weil's disease was characterized by a sudden onset, usually with chill, and always with high fever. This sudden and violent onset served as a valuable distinguishing sign between Weil's disease and typhoid fever with jaundice. In most cases delirium developed early, and albuminuria was a constant manifestation. Usually within eight or ten days the temperature would fall by lysis, and the other symptoms would become correspond-

ingly better. Relapses were not uncommon. The disease often occurred epidemically, but was apparently not contagious. It was most commonly observed in healthy male adults, and especially in Germans, French, and Russians. The disease was almost certainly the result of an infection, and the source was undoubtedly putrid animal flesh. It had long been known that butchers were very commonly affected, as were also those classes who consumed sausages largely. The speaker said that he had seen one case which had followed eating heartily of lobster salad. The disease was usually marked by congestion and swelling of the spleen and lymphatics, together with parenchymatous nephritis, swelling, and parenchymatous degeneration of the liver, and the presence of pronounced icterus. There was a marked resemblance between a fatal case of Weil's disease and acute yellow atrophy of the liver. The microscopic lesions were quite similar in the two conditions. Acute infectious icterus might be easily confounded with mild yellow fever. Bacteriological investigation of the disease had, for the most part, yielded negative results. Jaeger had, however, studied ten cases of the disease, three patients having died, two coming to autopsy. He had succeeded in isolating from these cases a special germ, and claimed that by inoculating it into mice he had produced lesions similar to those of Weil's disease.

Dr. BROOKS then made the following report of a case that had come under his own observation. The patient was an American laborer, thirty-three years of age, who gave no history of having eaten tainted meat, and who had been in good health up to six days prior to admission to the hospital. The disease had suddenly announced itself by fever and great pain in the muscles, and, three days later, there had been marked jaundice, with diarrhoea and nausea. In a short time there had been



marked delirium, and he had been comatose on admission to the service of Dr. Meltzer at the Harlem Hospital. At this time the jaundice was extreme, his temperature was 102° F., and the pulse full, strong, and slow; the urine contained albumin. He died the next day. At the autopsy the skin over the entire body was decidedly jaundiced; the pupils were widely dilated; the ankles were oedematous. All of the viscera were deeply bile-stained. The blood was fluid, and stained the hands a bright yellow. The liver was enlarged, its capsule was tense, smooth, and non-adherent, the tissue soft, and the lobules plainly marked. The entire liver tissue was stained with bile. The gall bladder was enlarged, and contained about thirty cubic centimetres of yellow, fluid bile. The duct was patent and normal. The liver weighed three and one-half pounds. The spleen was greatly enlarged; its capsule was tense and the tissue of a deep purple color. The mesenteric and retroperitoneal lymph nodes were enlarged and inflamed. The intestine was distended by gas, and contained a considerable quantity of soft, gray faeces. The kidneys were enlarged, their capsules were intimately adherent, and they weighed six ounces each. Microscopical examination of the cerebrum revealed a large number of degenerated ganglion cells, in a few instances with morphological destruction of both protoplasm and nucleus. The cerebellar cells also showed degeneration. The sections of the liver could hardly be recognized as liver tissue. The entire interlobular stroma showed extensive round-cell infiltration; this engorgement with small round cells extended even into the intralobular stroma. The most marked changes were in the liver cells themselves, and these changes could be divided into three well-defined classes, viz.: (1) Cells bordering on the larger capillaries of the portal system and medium-sized



bile ducts, which were almost completely destroyed; (2) groups of cells representing the centres of degenerated liver lobules; and (3) cells from the centres of the lobules, but more nearly normal than in the second class. The cells of the third group remained in the general form of the liver lobule. The protoplasm of these cells, though least involved of all, was very granular and contained large fat spaces. There were numerous micro-organisms found. On bacteriological examination of the liver and spleen, bacilli were found, and these were of two varieties, viz.: (1) A rather short bacillus with rounded ends, staining best with Loeffler's methylene blue, and having an irregular staining or mottled body; and (2) a variety differing from the first in only being thinner and longer. Enormous cocci (involution forms) were also present, but they were much less numerous than the bacilli. The latter morphologically resembled very closely diphtheria bacilli. It was concluded that the germ was a *Proteus*, and it was found pathogenic to guinea-pigs and mice. It resembled very closely that found by Jaeger in an epidemic. The post-mortem examinations on the inoculated animals showed the mucous membrane of the mouth to be yellowish, and there usually was clear yellowish fluid in the peritoneal cavity. The liver, kidneys, and spleen were always much congested. Microscopic examination of the liver and spleen revealed acute parenchymatous and fatty degeneration of the liver and kidneys. The bladder contained highly acid urine. The bacillus was obtained in pure culture from the peritoneal exudate, the liver, kidneys, spleen, and lymphatic nodes, and was usually absent in the blood. The cortical ganglion cells of the cerebrum and cerebellum, under Nissl's stain, showed very extensive degeneration. The older cultures were found more virulent than the fresh ones; hence it was probable that a

toxin was developed. Subsequent experiments demonstrated that the germ produces in culture media a toxin fatal to animals and producing the general lesions of the disease. Various attempts had been made to inoculate animals along the gastro-intestinal tract, but with negative results except in one case—a monkey. The pathological findings in this animal were similar to those in the guinea-pigs. Fatal and typical lesions were induced in a monkey by introducing the germ into the portal vein. Although jaundice was not produced in any of the experimental animals, the liver was always found to have undergone marked degeneration. The nephritis—one of the most important features of the disease—had been uniformly reproduced in the inoculated animals. Post-mortem changes in the tissues had been specially guarded against in the experimental studies. The delirium was probably the result of the degeneration of the ganglion cells. From the foregoing experiments, the speaker said, he did not feel like considering the bacillus a specific one of this disease. He believed that other germs gaining access to the system in the same manner might produce similar symptoms and changes. Weil's disease, acute yellow atrophy of the liver, yellow fever, and phosphorus poisoning were all conditions dependent upon toxæmia, bacteriologic or otherwise, the lesions found in all were similar, and the symptoms of each were practically identical; hence he said that he thought the pathogenesis in all four diseases was the same, although the specific exciting agent differed. The symptomatology was probably, at least partly, dependent on the extensive destruction of liver tissue.

#### *Discussion.*

Dr. E. LIBMAN said that he had recently observed a case of appendicitis in the second attack. Dr. H. Lili-



enthal had operated upon the abscess, and from the pus there had been obtained a pure culture of the bacillus proteus fluorescens. On the following day the patient had developed jaundice, tenderness of the liver, enlargement of the spleen, and albuminuria. After a few days these organs had returned to their normal size, and the patient had been convalescent. When Jaeger had described a large group of cases associated with the bacillus proteus fluorescens, it had seemed proper to call the disorder Weil's disease, but the case just reported would throw doubt upon the wisdom of such a nomenclature. It would be better to call these cases infections with the proteus bacillus than to speak of them as Weil's disease.

Dr. P. A. LEVENE said, regarding the action of the liver on toxins, that this organ was ordinarily supposed to neutralize toxins; yet, according to the description given in the paper, certain bacterial products seemed to have been more toxic after having passed the portal circulation than before.

---

*Stated Meeting, March 8, 1899.*

T. MITCHELL PRUDDEN, M.D., PRESIDENT.

TWO CASES OF GANGRENOUS BRONCHITIS WITH ISOLATION  
OF AN ORGANISM RELATED TO STREPTOTHRIX.

Drs. CHARLES NORRIS and J. H. LARKIN reported these cases. The first case was that of a man who had complained chiefly of cough and dyspnoea for a month and a half before coming into the hospital. He had died eight days after admission, evidently of sepsis, the temperature having ranged between 103° and 104° F. The clinical diagnosis had been probable gangrene of the lungs. The autopsy was made on January 23, by



Dr. Larkin, two hours after death. The left lung was found to be bound down by old adhesions, and the lymph nodes at the root of both lungs were extremely large. The mucous membrane of the trachea and bronchi was œdematous, and contained white granules, about the size of millet seed. On section, myriads of white foci were seen scattered over the lung, occluding the smaller bronchi. The lesions were confined to the bronchi. The odor of the lung was extremely fetid, and its color was dark green. The second case which they reported through the kindness of Dr. E. Hodenpyl, presented nothing of importance in the personal history. The patient had been perfectly well up to January 1st, but since then had suffered from cough, profuse and fetid expectoration, and pain in the chest. Examination showed flatness over the lower part of the right lung, with bronchial voice and breathing, and moist and crackling râles. The heart, liver, and spleen were normal. Death occurred on January 17th, and the autopsy was held twenty-four hours later. There was a fresh pleurisy over both lungs, and both showed extensive consolidation of the broncho-pneumonia type. The small bronchi contained pus and white granules, and the bronchial nodes were enlarged. The presence of opaque, whitish, and friable granules was the most noticeable feature. They were found in the secretion of the larger bronchi, or more often lightly adherent to the congested bronchial walls. The characteristic yellow color of the ray fungus of actinomycosis was absent. It was difficult to transfer the particles to the object glass. Under a low power they were seen to be made up of rods radiating toward the centre, the ends of these rods being often more or less swollen. No distinct bulbs or end capsules, so characteristic of actinomycosis, were observed. When stained with fuchsin one saw long, slender rods of irregular outline, as

well as rods slightly bulbous at their ends; cocci free or in short chains, but without characteristic grouping. Capsules could not be demonstrated by any of the usual methods. With Gram's staining all the cocci retained their color, but the rods were variable in their behavior. The plates, grown both aërobically and anaërobically on glycerin-agar and ascites serum, developed colonies very similar in appearance. Smears from these colonies showed short chains of cocci. Inoculations in mice were negative. There was no growth on potato. On gelatin a streptococcus appeared as whitish colonies along the punctures. With the material from the first case three intratracheal injections were given to rabbits, with negative results in two after two weeks. The third rabbit died later from a double empyema with adhesive pericarditis, the lungs being completely softened and necrotic. The empyemial pus showed large numbers of rods similar in appearance to those found in the granules of the bronchi and streptococci. Both these cases of gangrenous bronchitis presented similar clinical features and had run a fairly rapid course, characterized by fetid secretion and by the occurrence of whitish masses or grains, resembling those described under the name of pseudo-actinomycosis. The second case was the more acute. Sections of the lungs of both cases stained by Gram showed a peculiar, reddish, iodine reaction of the masses or grains. This reddish color of the streptothrix was also observed on the smears, and the same color reaction was obtained when stained by Gram's iodine solution. Absence of any growth except streptococci on the ordinary media at once separated both cases from the usual forms of gangrenous bronchitis. The fungus or streptothrix produced, in animals injected in the peritoneum, an adhesive peritonitis with localized collections of pus containing numerous typical rods, often with



extension of the inflammation to the pleura through the diaphragm. Intratracheal injections were followed by softening and necrosis of the lungs, with extension to the pericardium. Intravenous injection was also followed by necrosis and softening of the lungs with empyema. The marked predilection of the streptothrix to attack serous membranes, no matter how introduced, was an important feature. From the empyema of a rabbit injected intravenously with the grains of the second case, a growth on the cut surface of the rabbit's kidneys was obtained in the shape of translucent, whitish masses, resembling those seen in the bronchi of both cases. By continuous transplantation from kidney to kidney, they hoped to obtain a pure culture of the streptothrix free from streptococci. Distinct and positive branching has not yet been noticed. The rods presented interesting staining reactions in every respect. They were not dissimilar to the diphtheria bacilli. Metachromatic particles were especially noticed, staining deeply by hæmatoxylin, by Gram's stain, methyl blue, and by Ernst's stain.

Dr. J. H. LARKIN demonstrated the gross and microscopical specimens from the foregoing cases.

#### *Discussion.*

Dr. G. A. TUTTLE said that he had seen one case of streptothrix which agreed fairly well with the cases reported from Baltimore, but the lesions in his case were almost identical with those of tuberculosis, and the organism was very different from that just described. He now had it under cultivation, and found that it produced a lesion very similar to that of tuberculosis in animals.

Dr. LARKIN remarked that the brown color which the bacteria took in staining the sections with Gram was not permanent and it disappeared after about five days.



## DIFFUSE CARCINOMA OF THE STOMACH.

Dr. J. H. LARKIN presented specimens taken from a man, sixty-five years of age, who had entered the hospital during the very cold weather without any special history. He had complained of slight pain in the stomach, and had seemed quite weak. Examination of the abdomen had revealed a large tumor in the epigastrium and right and left hypochondriac regions. Two weeks after entering the hospital he died of exhaustion. At the autopsy it was found that the cavity of the stomach was very small, and that, with the exception of a portion of the lower curvature, it was extensively infiltrated with carcinoma. No metastases were found in the liver, but the spleen had been converted into a mass of carcinoma. The carcinoma had also involved the splenic flexure of the colon, producing partial stenosis. The most interesting feature was the escape of the liver and the total involvement of the spleen by carcinoma.

Dr. LARKIN then showed specimens from a case of

## HEMORRHAGIC PANCREATITIS WITH FAT NECROSIS.

The patient, a woman, fifty-six years of age, gave a history of indigestion lasting a year. On January 29th, about twelve hours after a hearty meal, she had had a severe attack of epigastric pain accompanied by vomiting. After a few days she had been able to go around, and had remained well until February 12th, when, about eight hours after a meal, severe and persistent epigastric pain, accompanied by profuse vomiting developed. Tympanites also developed quite rapidly. The bowels were obstinately constipated. At the autopsy the abdomen was found filled with fluid and coagulated blood. The heart, lungs, kidneys, and liver were normal. The pancreas was involved as a whole. It was greatly

increased in bulk, and about four times the normal size. It was of a dark red color, with small islands of necrotic lobules scattered throughout, bright yellow in color. There was extensive hemorrhagic infiltration of the pancreas. The intralobular fat tissue was necrotic, and presented similar appearances to the necrosis in the mesenteric fat tissue. There was diffuse hemorrhagic infiltration of the peripancreatic fat tissue, which was also necrotic.

A CASE OF SUPPURATIVE PANCREATITIS WITH RUPTURE  
OF THE PANCREATIC DUCT INTO THE STOMACH.

Dr. LARKIN reported this case, occurring in a woman, thirty-five years of age, who had been sick for one year with cirrhosis of the liver. Five months before death she had begun to get slightly jaundiced, and soon afterward there had been recurrent attacks of pain and vomiting, corresponding in every way with those of biliary colic, and recurring every fortnight. After about fourteen of these she had come to the hospital, and had had a severe attack there, followed in two weeks by a second attack, and one week later by a third. The patient died in great agony. At the autopsy the peritoneum posteriorly and down on to the kidneys was filled with old and recently coagulated blood. The heart, lungs, kidneys, and spleen were normal. The liver was cirrhotic. The pancreas was found to be very small and hard, and on cross-section the pancreatic duct was found to be an irregular and tortuous canal, which had become necrotic. There were no gall stones and the bile duct was pervious. On the lesser curvature of the stomach and posterior wall was a small, blackish area, in the centre of which were two small openings, which communicated directly with the pancreatic duct. The stomach was filled with black fluid, looking like digested blood.

A CASE OF SUPPURATIVE PANCREATITIS WITH ABSCESS OF  
THE STOMACH WALL, INVOLVEMENT OF THE  
SPLEEN, AND FATAL HEMORRHAGE.

Dr. LARKIN reported another case of suppurative pancreatitis, and presented the specimens, which had been taken from a man, fifty-six years of age, who had complained chiefly of pain for several months. A few days before entering the hospital he had developed jaundice. On admission the patient was jaundiced, and complained of abdominal pain and tenderness, and the abdomen was tympanitic. He continued to do badly, and died eight days after admission, no positive diagnosis having been made. The autopsy had been made on the following day. The abdomen was slightly tympanitic, and the peritoneal cavity filled with fluid and clotted blood. There was a large blood clot over the spleen. On the lower border of this organ was a large, ragged opening, through which the fatal hemorrhage had occurred. At the hilus of the spleen was a small abscess which passed directly through the pancreas. The pancreatic duct was dilated, and pus and detritus could be squeezed out. At the head of the pancreas was a very early carcinoma. In the greater curvature of the stomach was a large mass bulging into the stomach. The mucous membrane over it was normal. It proved to be an abscess in the wall of the stomach, which had formed in the serosa proper. There was no connection between the pancreas and this abscess.

*Discussion.*

Dr. G. A. TUTTLE said that in his experience hemorrhagic pancreatitis had always been a disease of the blood-vessels, and hence it did not seem to him proper



to speak of these cases as examples of hemorrhagic pancreatitis, any more than to call a cerebral apoplexy a hemorrhagic cerebritis.

Dr. F. C. WOOD remarked in this connection that in one of the cases there had been a marked fat necrosis, showing that there was something more than a simple apoplexy.

Dr. W. H. PARK said that two diphtheria antitoxin horses belonging to the Health Department had suddenly died, and at autopsy there had been found a large quantity of blood in the peritoneal cavity and in the liver. The quantity of blood in the liver was about four times the weight of this organ. As yet there had been no report on the condition of the arteries.

#### A CASE OF MULTIPLE FIBROMA.

Dr. HARLOW BROOKS presented the fresh specimens from the case of Mike Kelly, seventy-seven years of age, who had been for twenty-five years in Bellevue Hospital. The old man had been going about the wards as usual, when he became unconscious and died within a few hours. The sudden death was explained by the lesions found at autopsy. The most prominent and best-known lesion in this famous case—the fibroma of the scalp, which hung down on the right side of the head—had been completely removed, and was presented with other specimens. It had not been adherent to the bone, but only to the superficial layers of the scalp, and the periosteum was only slightly thickened over this area. Over the skin were numerous nodules looking like papillomata on superficial examination, but closer inspection showed them to be elastic, and situated entirely beneath the epithelium in the subcutaneous tissues. These numerous fibromatous masses were of varying size. They presented no symmetry in their distribution,

and had no apparent connection with the nerve trunks. The nodules somewhat resembled lipomata, but were much firmer. All the nodules, including the one over the crest of the ileum, were encapsulated. In the upper part of the œsophagus were found a few, small, apparently fibromatous masses beneath the mucosa of the upper portion. The heart and lungs were negative. The mucous membrane of the stomach was extremely atrophic. Beneath the peritoneal surface of the stomach were numerous nodules, measuring about three or four millimetres in diameter, and apparently encapsulated. In the duodenum, beneath the peritoneum, easily dissected out without injury to the mucosa, and covered by endothelium but not by connective tissue, were nodules of jet-black color. On section they did not present the appearance of fibromata, and were apparently not lymph nodes. In the ileum only a very few of these nodules were found, and there was none in the walls of the caput coli or colon. The caput coli was carried over to the median line, and was adherent to the right side of the lumbar portion of the spinal column. The appendix lay directly behind, and was adherent to the posterior surface of the caput. At about the level of the crest of the left ileum there was a complete fold in the intestines, which passed upward to the coeliac axis, where it was adherent to the duodenum. This peculiar arrangement of the intestines recalled the fact that at times this man had presented symptoms of intestinal obstruction. Careful search had been made throughout the body, but no evidence of tubercle had been found, though the patient had been resident in the hospital for years. The cause of death was found to be acute nephritis. There was great œdema of the brain and of its membranes. A report of the microscopic examination of the tumors was promised for the next session of the Society.

## PULMONARY VALVE WITH FOUR CUSPS.

Dr. LEON T. LEWALD presented a heart showing four cusps on the pulmonary valve, three of about equal size, and one very much smaller. The specimen had been taken from a man, thirty-two years of age, who had died of pneumonia.

## COMPLETE TRANSPOSITION OF ALL THE VISCERA.

Dr. LEWALD also showed specimens from this case. They had been removed from a woman, twenty-eight years of age, a native of the West Indies, who had died from an acute lobar pneumonia "on the right side, but in the left lung." The heart was turned upon itself so that the aorta was in front. The heart was practically normal. The arteries normally coming from the right side of the aorta were given off from the left side, and *vice versa*. The lung on the left side had three lobes, while that on the right had two lobes. The suprarenal bodies were transposed.

## ABSCESS OF THE LIVER DISCHARGING INTO THE LUNG.

Dr. LEWALD then exhibited some post-mortem specimens just removed from a man, forty-five years of age. The autopsy showed an abscess of the liver, probably amoebic, containing fetid pus. It had penetrated the diaphragm and had discharged into the lower lobe of the right lung. There was some ulceration of the cæcum.

## AN ADENOMA OF THE THYROID GLAND.

Dr. LEWALD further showed a tumor of the thyroid gland, taken from a man, thirty-one years of age, who died of pulmonary tuberculosis. There had been no symptoms referable to the thyroid, although the



tumor was of considerable size. Microscopical examination showed it to be an adenoma. There was no metastatic growth or further involvement.

Dr. FRANCIS C. WOOD then read a paper on

PUERPERAL INFECTION WITH THE BACILLUS AËROGENES  
CAPSULATUS.

During the six years which have elapsed since the publication by Welch and Nuttall of a case of terminal infection with a peculiar gas-forming bacillus, there have been reported in the literature a considerable number of such cases, varying greatly in the time and portal of infection and in the clinical picture of the disease. The following patient entered St. Luke's Hospital, in the service of Dr. G. A. Spaulding, to whom I am indebted for the clinical notes.

The patient on admission was so ill that a consecutive history was obtained with some difficulty. She said that she had always enjoyed good health up to the present time, and had borne eleven children. Before the birth of the last a considerable oedema of the lower extremities had been noticed, but no other evidence of renal disease and no eclamptic seizures. About two weeks before admission she began to feel weak and had some dyspnoea and a slight cough, and two days before she noticed that her face had begun to swell. When examined on admission she was found to be a large woman with abundant subcutaneous fat. There was a moderate oedema of the face and limbs. The heart was enlarged, the apex beat being six inches from the median line. The action was rapid and forcible, with a marked accentuation of the aortic second sound. The pulse was quite tense. An enlarged uterus occupied the lower part of the abdomen. The foetal heart sounds could not be heard. The temperature on admission was 102° F.;

pulse, 108; respiration, 32. The urine was acid; specific gravity, 1.020. It contained so much albumin that it solidified on boiling, and there were abundant granular casts and some blood found on microscopical examination. The amount of urea was greatly diminished.

Under these conditions it seemed proper to remove the foetus, and accordingly a sterilized soft-rubber catheter was passed to the fundus of the uterus and left in place for twelve hours. After digital dilatation of the cervix, the foetus was expelled twenty-four hours after the passage of the catheter. It was apparently about six months old, and was not macerated. Soon after the delivery of the patient a manual examination of the interior of the uterus was made, in order to find, if possible, a cause for a rather severe post-partum hemorrhage, and to be sure that all placental tissue had been removed. The temperature on the day of delivery reached 103.4° F., with a pulse of 120 and respiration 40. The patient was drowsy and stupid. The urine still contained large quantities of albumin and many casts. The abdomen was somewhat distended. About this time it was noticed that the lochial discharge was very offensive, so much so that it was necessary to remove the patient from the ward to a separate room, because of the annoyance to those next to her. The following day the temperature remained continuously above 102° F., while the pulse and respiration increased in frequency and the abdominal distention was very marked. No subcutaneous oedema was noticed. She died the third day after delivery.

An autopsy was made twenty-four hours after death, the body having been kept on ice in the meantime. The more important points are as follows: The whole body was noticeably swollen and there was a marked emphysema of the subcutaneous tissues. The superficial veins



were distended with gas, and on opening the abdomen a large amount of gas escaped. The intestines were of a deep red from dissolved blood pigment, and the peritoneal coat was dull and covered with flakes of fibrin. Five hundred cubic centimetres of bloody fluid were found free in the dependent parts of the cavity. On opening the pericardium some gas escaped, and a fresh pericarditis was present with a moderate amount of fibrin. The heart chambers were empty and the muscle contained gas bubbles. There was a long branching thrombus in the pulmonary artery of the left side, with an infarct in the lower part of the upper lobe. The liver was slightly enlarged, and its substance was converted into a meshwork by the formation of large and small gas bubbles. There was no fluid or foam exuding from the cut surface; on the contrary, the liver tissue was dry and of a dull opaque red, as if it had been cooked. The spleen was not enlarged and contained numerous gas bubbles. The kidneys were large; the capsule stripped easily; the substance was œdematous and rotten. Gas could be squeezed from the vessels. No markings could be made out. The uterus and vagina were lined with a foul-smelling, blackish slough, very soft and pulpy, and about 5-15 mm. thick. There was no perforation or tear in the uterus or vagina. Smears from the uterus showed enormous numbers of the capsulated rods, a very few streptococci in short chains, and numerous short bacilli, probably colon. Smears from the other organs and the pericardial and peritoneal fluids showed only large, thick, encapsulated rods, positive to Gram. Cultures from the uterus grown at 37° C., with free access of air, showed only colon bacilli. Cultures from the other organs and the pericardium under similar conditions gave no growth. Anaërobic cultures, on the other hand, furnished an abundant growth of bacilli, which corresponded



in every particular, including pathogenic properties toward animals, with the bacillus described by Welch and Nuttall under the name of bacillus aërogenes capsulatus. For any morphological or cultural details, the reader is referred to their paper in the *Johns Hopkins Hospital Bulletin* for 1892.

Microscopic sections of the viscera showed some interesting points: The uterine mucosa had entirely disappeared and also the placental remains. In the necrotic layer lining the uterus and vagina were an enormous number of bacteria. First and most prominent with a Gram stain were the large rods of the gas bacillus. Besides these were a large number of what were probably colon bacilli and a few streptococci and diplococci. These cocci could not be isolated on culture media. In sections toward the peritoneal surface of the uterus the vessels, especially the veins, were filled with thrombi which contained only the gas bacillus; this form was also found in the uterine lymphatics, so that the streptococcus invasion did not extend deeply. The liver stained very poorly; most of the cells showed a very marked degeneration, which was especially noticeable in the tissue surrounding the gas bubbles. The walls of the latter were crowded with enormous numbers of the bacilli. In the kidneys, besides a very advanced chronic diffuse nephritis, there were many gas bubbles, and the bacilli in this case had also entered the glomeruli and the tubules, and could be seen on the surface of the large casts which still remained in the tubules. The heart muscle, the pericardial fibrin, the pulmonary thrombus, and the lungs all contained an enormous number of the characteristic bacilli. Sections from the hairy scalp of the foetus showed a large number of the same large bacilli, positive to Gram, both on the surface and in the lymph spaces of the corium. Unfortunately the foetus was placed in al-

cohol immediately after delivery, so that no cultures could be obtained, nor were cultures made from the lochial discharge during life.

A number of questions arise in the interpretation of this case, which are difficult to answer in any absolute manner. The first and perhaps the most important is the determination of the time of the infection. The wide distribution of the bacilli throughout the body is in no way incompatible with an infection immediately ante-mortem, for if a guinea-pig is injected subcutaneously with a virulent culture, and then in a few hours is killed and kept in a warm place, the body will soon swell because of the gas production and be found to be quite generally invaded by the bacilli, which multiply with enormous rapidity in the blood, and, as the gas is formed, are pushed along the vessels into the capillaries and thus invade the parenchyma of the organs. The importance of finding the bacilli in the subcutaneous tissues of the child was very slight as regards the determination of the time of infection, for without cultural results it became a mere probability that the forms found were the specific bacillus. On the other hand, two cases of ante-partum infection of the child and amniotic fluids have been reported, though in these cases the mother did not die. It is possible, therefore, that the infection took place at the time of the production of the abortion, and that the placental tissues offered a favorable site for infection. The result of the microscopical examination of the liver and kidneys rather pointed to an infection some time before death, for the cells of these two organs were in a condition of extreme degeneration, most marked about the walls of the air bubbles. This is said not to take place when the bubbles are formed after death. In the heart muscle also, there were areas of inflamma-



tory reaction around the gas bubbles, a process that could not have occurred within a short time of death. The exact part played by the streptococci found in the slough lining the uterine cavity is doubtful. The number found was exceedingly small, and none was found in the lymphatics of the wall or peritoneal surface of the uterus, as is usually the case in any severe post-partum infection with this species. It seems probable that both the streptococcus and the colon bacillus can be disregarded in this case.

From a clinical aspect the whole course of the disease changed from the day of delivery. The temperature became continuously high, the abdomen began to swell and was tympanitic, yet there was no intestinal distention on autopsy; only a large quantity of gas free in the peritoneal cavity. The odor of the lochial discharge was noticed soon after delivery, and resembled that of the gas escaping from the peritoneum.

#### *Discussion.*

Dr. W. H. PARK suggested that a period of four or five days was too long to justify the assumption that the gas bacillus was the cause of death. With the presence of the streptococcus and colon bacillus infection, there was plenty of reason for the illness.

Dr. Wood replied that the case had been complicated with nephritis and a high temperature. The period of infection with the gas bacillus he would limit to two days before death.

Dr. E. K. DUNHAM said that his cases of undoubted ante-mortem infection with the gas bacillus had been, for the most part, operative cases, with infection in the urethra. In one, the temperature had risen within a very few hours after the operation, and had reached 106° or 107° F. before death, which occurred at the



end of about thirty-six hours. The gas bacillus was present in great abundance, and was found even before death. In all but one of his cases the presence of the gas bacillus before death had been demonstrated.

Dr. HARLOW BROOKS said that there had been two cases at one of the hospitals in which the infection seemed to have occurred after death. He had made the autopsy in one case about four hours after death, and in this one the gas had already begun to form. This patient had fallen out of a window while menstruating, and the vagina had been filled with blood. Infection seemed to have taken place after death, as no clinical manifestations of it were present and it was not suspected until the autopsy was begun. Infection in this case was thought to have taken place through a traumatic tear in the wall of the rectum, which extended nearly through the walls of the vagina.

---

*Stated Meeting, April 12, 1899.*

T. MITCHELL PRUDDEN, M.D., PRESIDENT.

A CASE OF HEMIATROPHY OF THE BRAIN OF A CHILD.

Dr. DAVID BOVAIRD exhibited the brain from a child, eleven months old. The infant had been returned to the New York Foundling Asylum suffering from severe diarrhoea, and this had been the cause of death. During the five days it had been under observation the asymmetry of the skull had been noted, together with the fact that the child was exceedingly restless, and was prone to beat its head against the crib. The child had made no attempt at walking or talking. There was no paralysis. The autopsy had revealed on the inner surface of the dura mater over the left half of the brain a pad of fibrous tissue, well supplied with blood-vessels.

It was closely adherent to the dura and could be separated from the pia only by tearing. Examination of the brain had shown marked atrophy of nearly the whole of the left hemisphere. The lesion was confined to that portion of the brain supplied by the middle cerebral artery. The tip of the front lobe, the occipital, and lower portions of the temporo-sphenoidal were normal. The median surface was normal. Throughout the affected area the convolutions were shrunken and small, and the sulci were widened. The withered convolutions were much harder than normal, and of a peculiar yellow wax-like color. Although the distribution of the atrophy suggested a lesion of the middle cerebral artery, that vessel was apparently intact and pervious. The mat of fibrous tissue lining the dura over the atrophic hemisphere was considered a secondary and complementary lesion, the primary process being the atrophy of the brain.

ACUTE MENINGO-ENCEPHALITIS WITH ŒDEMA OF THE  
CEREBRUM OF THE AFFECTED SIDE SIMU-  
LATING HEMIATROPHY.

Dr. EUGENE HODENPYL presented in connection with this case a specimen from a case of acute meningo-encephalitis simulating hemiatrophy of the brain. The specimen had been taken from a man who had been found unconscious in the street, and who had died a few hours after entering the hospital. At the autopsy a purulent otitis media had been found on the right side. The right hemisphere was considerably larger than the left, and the pia on that side was thickly covered with an exudate of fibrin and pus. Microscopical examination of the brain of the right side showed that its increased size and increased softened consistence were due to a well-marked œdema.



A CASE OF OCCLUSION OF THE CORONARY ARTERY, WITH  
INFARCTION OF THE MYOCARDIUM, AND SUDDEN DEATH.

Dr. F. C. Wood then showed a specimen of infarction of the heart muscle causing death with symptoms of angina pectoris. The patient had been a well-nourished, previously healthy man of about fifty-five years of age. For some six weeks before his death he had suffered from a rather acute bronchitis, but this had become so greatly improved that he had been able to return to his office, though still troubled by an occasional severe pain in the left chest. The next day he was distinctly worse, and died within twenty-four hours, with very great pain over the præcordium, radiating down the arm, and with a feeling of impending death. The heart action was rapid and feeble. The autopsy showed a moderate chronic nephritis and a thrombosis of the anterior branch of the left coronary artery, which was very atheromatous, as was also the right. The point of obstruction of the vessel was about three centimetres from its origin in the aorta, just after the giving off of the septal branch. Here there was a firm thrombus attached to the wall of the vessel. The heart muscle was changed over an irregular area, measuring about three by two centimetres. It was of a pale, opaque yellow, very friable and soft. The softening and change did not extend the whole thickness of the ventricular wall, there being at least half a centimetre of healthy muscle between it and the ventricular cavity. Microscopically the myocardium was necrosed and fatty, with a considerable infiltration by an inflammatory exudate. No bacteria could be demonstrated by staining. The condition represented quite accurately the results obtained in animals after ligaturing the coronary for twenty-four to thirty-six hours, so it might be presumed that the patient's severe



symptoms were due to the beginning changes in the myocardium. A partial expression of the irregular cardiac contractions before death was shown by the presence of a very well-marked fragmentation of the myocardium. Such cases of thrombosis were not uncommon, but the much more usual course was for the obliterating process of the endarteritis to take place so slowly that the necrosed areas were replaced by fibrous tissue, without any very marked symptoms to call attention to the change.

A CASE OF SUBACUTE MYOMALACIA FOLLOWING SEPTIC  
INFLAMMATION OF THE LEFT URETER.

Dr. CARLIN PHILLIPS presented a heart that had been taken from a man, fifty-four years of age, a laborer, who had been admitted to Bellevue Hospital on April 18, 1899. He was alcoholic in habits, but denied syphilitic infection. He had had the ordinary diseases of childhood, and at the age of twenty-four years had smallpox. Since the age of five years he had had weekly attacks of renal colic. In 1894 he had had a nephrectomy performed for renal calculi. Since that time he had been free from attacks of great abdominal pain. One week before admission the patient complained of a severe cold, with pain over the upper portion of the sternum, and constant cough. He said the pain was not very severe, but it gradually increased. He had chilly sensations at times, coming on irregularly and without definite rigor. The patient had been in bed part of the time, and was extremely sensitive to cold. He had some nausea and headache, but no vomiting. He had good appetite, but was sleepless. He entered the hospital on account of cough, weakness, and pain in the chest. On admission, his pulse was 80; respiration, 22; temperature, 100.4° F. The heart dulness was increased. The apex

was at the sixth intercostal space and one-half an inch outside of the nipple line. The sounds were weak. No murmurs were present. There were fine moist râles over both lungs posteriorly, and broncho-vesicular breathing over the right apex; fine friction sounds and subcrepitant râles were heard over the right infraclavicular region. The epigastrium was tender but not distended, and was everywhere tympanitic on percussion. The liver dullness was somewhat increased. The patient's temperature was between 99° and 100° F. most of the time, but during the second and last week it reached 102° F. on two occasions. He suffered from great pain in the abdomen and also from pain over the upper portion of the sternum. The pulse varied between 110 and 120, and occasionally reached 130. The urine had specific gravity 1.031; it was free from blood, albumin, etc., but contained a few hyaline and granular casts. The patient died on February 3d, or about two weeks after admission. The clinical diagnosis was acute myocarditis and peritonitis.

The autopsy was performed by Drs. McAlpin and LeWald two days later. The findings in brief were acute bronchitis, hypostatic congestion and oedema of the lungs, chronic adhesive peritonitis. The region of the left kidney was filled with a large vascularized cicatrix of connective tissue and fat. The left ureter was found as a fibrous cord with obliterated lumen. Along the ureter were found a few minute pockets of greenish pus. The right kidney was enlarged, and showed large recent hemorrhagic infarctions. On opening the pericardium it was seen to contain about six ounces of fluid blood. The heart was enlarged. In the region of the left apex was an aneurismal dilatation, measuring 5 cm. in diameter. This area was peculiarly dark, with fine yellow mottling. On opening the ventricles there was seen to



be a dark grumous clot partially filling the aneurismal pouch, and intimately adherent to the contiguous wall. The valves were but slightly thickened and perfectly competent, and the remaining endocardium was normal. The anterior coronary artery was completely thrombosed throughout, but showed only slight atheromatous changes. Microscopically, the coronary vessels were seen to contain an old parietal thrombus, partially organized, not obliterating the lumen, and a fresh thrombus completely occluding the vessel. The heart wall in the affected regions showed microscopically the greatest multiplicity of changes, which differed from the usual picture seen in acute infarctions of the heart. In brief, there were organizing thrombus in the ventricle, simple atrophy, brown atrophy, fatty degeneration, simple necrosis, Zenker's degeneration, cloudy swelling, areas of purulent foci, and subacute interstitial myocarditis. Owing to the absence of pronounced atheromatous changes in any of the vessels, and especially of the coronary arteries, the findings in the heart were considered to be the result of septic thrombosis following infection from the left ureter.

#### *Discussion.*

Dr. PHILLIPS said that Birch-Hirschfeld stated that this condition was of extreme rarity, and for that reason the specimen was presented.

#### A CASE OF HEMORRHAGIC INFILTRATION OF THE MYOCARDIUM WITH INTERSTITIAL MYOCARDITIS.

Dr. J. H. LARKIN presented specimens from a case of hemorrhagic infarction of the heart. They had been taken from a man, thirty-seven years of age, who was admitted to Bellevue Hospital, January 16, 1897, with the following history: He had always been in good



health till three weeks ago. He never had rheumatism; did not use beer or whiskey. Four weeks ago, in December 18, 1896, he caught cold but kept on working. For a week he was troubled with severe pain about the heart; the pain was not made worse by inspiration; it was of a lancing character, not constant. The pain still continued, but was not so severe as at first. On December 25th, three weeks ago, he had to stop work on account of shortness of breath and feeling sick generally. Dyspnœa, which was at first noticed a month ago, had gradually increased. He had had orthopnœa for three weeks. He had cough with profuse expectoration. On January 14th, he raised about one-half an ounce of blood. His feet were swollen. His temperature was 100° F.; pulse, 116; respiration, 28. On examination, the heart sounds were feeble and rapid. There was no murmur. The heart was enlarged; the impulse was in the axillary line, sixth interspace; the sounds were more distinct at this point. There were pleuritic râles over the cardiac region, and the right base posteriorly. The pulse was small, weak, and rapid. When lying down there was pulsation of the vessels of the neck up to the lobe of the ear; when he stood erect, only half-way up, a harsh, short, diastolic murmur, most marked over the sternum, developed. On January 29th the patient was out of bed three times during the night. On the following morning, while sitting up in bed, he became unconscious, with twitchings of the entire half of the body, including the neck muscles but excluding the facial ones. There were yawning and lateral nystagmus, with diminution of the pupil of left eye. He had Cheyne-Stokes breathing. He died shortly afterward. At the autopsy nothing abnormal was noticed in the brain. The lungs were normal. The heart was slightly increased in size. There was no valvular lesion. In the wall of the left ventricle

was a dark area, which contrasted sharply with the surrounding pale heart muscle, extending from the endocardial surface to about 3 mm. from the pericardial surface. The cardiac muscle in this area was dark and quite soft and depressed below surrounding muscle. A cross-section showed the darkened area to be about 2 cm. long. The spleen was large and congested. The liver was in a state of chronic venous congestion. The kidneys showed a large congested surface; granular markings were not evident. Microscopical examination of the heart showed that the muscle in the infarcted area had been replaced by hemorrhagic extravasation. Scattered through this blood clot were remnants of dead heart-muscle cells, without nuclei, many containing larger and smaller vacuoles. At the periphery of the area was a narrow band of newly formed fibrous tissue, with thin-walled blood-vessels and oedematous stroma. In places this tissue had proliferated between the muscle fibres so that it made a solid mass; and aside from the lesion proper it looked not unlike spindle-cell sarcoma. Changes in the coronary artery from the same case showed extensive obliterating endarteritis. There was great thickening of intima, with secondary degeneration and calcification.

Dr. LARKIN also presented specimens from

#### A CASE OF INTERSTITIAL MYOCARDITIS FOLLOWING SCARLATINA AND DIPHTHERIA.

The patient was a girl of fifteen years, who had always been well till December, 1896, when she had diphtheria and scarlet fever, from which she apparently recovered. Early in January, 1897, she began to suffer from marked dyspnoea, and was brought to Roosevelt Hospital on January 10th. On admission she was



well nourished; somewhat pale; with bounding and irregular pulse. There were dyspnoea and oedema of both lower extremities. Urine: specific gravity, 1.012; containing twenty per cent. albumin, with granular and hyaline cysts. The lungs were normal. The heart was increased in size, and there was a double murmur at the apex and base. She continued to do badly until January 13th, when she died comatose.

An autopsy was made on January 14th by Dr. Ewing. The lungs were oedematous, otherwise normal. The left ventricle of the heart was hypertrophied; there were soft, fresh vegetations on the aortic and mitral valves and on the surface of the endocardium of the left ventricle. About midway between the base and apex on the anterior left lateral half of the left ventricle the cardiac muscle was dark and somewhat soft. This area occupied the inner two-thirds of the heart muscle; the outer third appeared normal; the area extended vertically about one inch. The liver was pale and fatty. The spleen was hard and congested. The kidneys showed acute nephritis, with marked congestion of the pyramids, and a fine granular surface. The microscopical examination showed that a large area of the heart muscle was replaced by a new-formed connective tissue, in part very cellular, in part fibrous. This tissue formed in one place a solid mass; in other places it lay between the muscle fibres, which showed various phases of atrophy and degeneration. The new-formed connective tissue was noteworthy on account of the very numerous thin-walled blood-vessels, which in places were dilated and had evidently ruptured, giving rise to interstitial hemorrhages. The kidneys showed subacute diffuse nephritis, with marked oedema of interstitial tissue; there was thickening of Bowman's membrane, with increase of cells in and on the capillary tufts; exudate was found in the tubules,



with parenchymatous changes in the epithelium and congestion of the blood-vessels. There was moderate fatty infiltration of the liver; the cells stained poorly; numerous areas of focal necrosis were seen similar to those found in typhoid. These areas of necrosis were especially numerous in the spleen, being much larger than those in the liver. To this there was added chronic congestion of the splenic pulp.

As to the possible cause of the myocarditis in this case, a number of factors were to be thought of. Inasmuch as there was an acute endocarditis with soft fresh vegetations on the aortic and mitral valves, these, if detached, might serve as emboli to the coronary artery or its branches, thereby shutting off the blood supply from a given area of heart muscle with resulting infarction and subsequent replacement by fibrous tissue. But embolism of the coronary artery was least probable, as the mechanical action of the heart in systole drives the bulk of the blood into the aorta past the coronary orifice, and as the position of the artery was at right angles to the current of blood in the aorta. Embolism of this artery is, however, a possibility. The existence of focal necrosis in other viscera in this case, following scarlatina and diphtheria, would suggest the possibility of a similar origin of the primary heart lesion. The specimen was especially interesting on account of the over-production of blood-vessels and the secondary interstitial hemorrhage in an area of new-formed connective tissue, which was evidently in the way of restoring as well as might be the integrity of the heart.

A CASE OF CUTANEOUS SARCOMA OF THE LEG FOLLOWING  
A BURN.

Dr. L. T. LEWALD presented this case. The specimen had been removed by amputation from a woman,

twenty-nine years of age. She received a burn twenty-four years ago, and the area never completely healed, there having been a granulating surface and two deep discharging sinuses. About two years ago an attempt was made to excise the sinuses and secure healing, but the operation failed, and slough occurred. During the last four months the ulcer took on a fungous growth. Microscopical examination of a small section of the tumor seemed to warrant amputation, and this was done about five weeks ago. Since that time an operation was performed on the enlarged glands of the inguinal and femoral regions. They were involved in the malignant process.

*Discussion.*

Dr. E. K. DUNHAM exhibited under the microscope a series of sections made through the fungous mass. They showed what appeared to be a rather large spindle-cell sarcoma, and the same structure, he said, had been found in the supposed metastatic growth. The section from the ulcerated portion that had not become fungous showed an apparently typical picture of an epithelioma. It might be an accidental appearance, however, resulting from the coincident attempt of the epithelium to cover the granulating surface. The speaker asserted that when proliferation had been going on so long, one would ordinarily hesitate to say positively, from an examination of a portion of tissue from the granulating surface, that the case was or was not a sarcoma. In this instance, however, there seemed to be no room for doubt.

CUTANEOUS SARCOMA FOLLOWING A WASP-STING.

Dr. HODENPYL presented in this connection two cases of cutaneous sarcoma that had developed after a sting of a wasp. The first case was a spindle-cell sarcoma



that had developed over the sternum in a man, forty-six years of age, immediately after being stung by a wasp. This tumor had grown steadily until at the end of two years it had been removed by operation. So far there had been no recurrence. The second tumor had been removed from a man, sixty-three years of age, who had been stung in the thumb by a wasp. Immediately afterward a nodule had developed on this part, and the tumor had been excised at the end of three months. Microscopical examination had shown the growth to be a small spindle-cell sarcoma.

#### A MALE PSEUDO-HERMAPHRODITE.

Dr. F. S. MATHEWS showed under the microscope sections of a testis taken from a supposed female child. At time of removal of the testis, January, 1892, the child was twelve years old. The operation was performed by Dr. Charles T. Poore at St. Mary's Hospital for Children. At the time of operation, the testis, which lay under the skin of the left thigh just below the external abdominal ring, was thought to be an enlarged lymph node. No microscopical examination was made until the present winter. The individual from whom the testis was removed was located after considerable search, and an examination permitted. The pseudo-hermaphrodite, now nineteen years of age, was tall, slim, and pale. The external genitals were those of a normal female. There was no hair on face or pubes. The vagina was one and one-quarter inches long. Through the vagina and rectum a very thorough pelvic examination could be made. Examination revealed no trace of uterus or prostate gland, nor was another testis to be found in the pelvis, iliac region, or outside the external abdominal ring. This individual then was a male pseudo-hermaphrodite. Most of the cases



described were either, like this one, undeveloped males, or males possessing well-developed male organs, and in addition vagina or uterus. No true hermaphrodite could be found described in the literature of the subject.

A paper by Dr. EUGENE HODENPYL was read—Subject:

MILIARY TUBERCULOSIS OF THE PLEURA WITHOUT OTHER  
TUBERCULOUS INVOLVEMENT OF THE LUNG.

If the pleural surfaces of adults are critically scrutinized after death, they will be found, not infrequently, more or less thickly studded with tiny white circumscribed nodules or patches. The real nature of these little nodules has not yet, it seems, been clearly defined. From a study which I have made of a large number of cases, I am of the opinion that they are neither simple fibromata nor fibrous hyperplasias, the result of coal-dust pigment, as is so commonly believed, but are in most instances miliary tubercles.

The object of this paper is, first, to draw attention to the relative frequency of miliary tuberculosis of the pleura without tuberculosis of the parenchyma of the lung, or so-called initial tuberculosis of the pleura; second, to point out that in this situation, perhaps more than in any other of the body, tubercles are prone to undergo more or less complete spontaneous healing; and, third, to indicate the probable significant relationship between this lesion and acute tuberculous pleurisy.

It is necessary to point out, at the outset, that tuberculosis may affect the pleura in one of two ways, either of which may be primary or secondary. First, the pleura is more or less thickly beset with nodules and patches, which are the result of a tuberculous inflammation (miliary tuberculosis). Second, in addition to

the above, there may be an escape of living germs into the pleural cavity, with the result that an acute exudative inflammation, with the production of serum, fibrin, and pus, is developed. This latter condition is usually spoken of as acute tuberculous pleurisy.

While primary miliary tuberculosis has for a long time been recognized as an independent pathological lesion, having been mentioned as far back as 1827, the subject has, nevertheless, so far as I can ascertain, from the literature at hand, received but scant attention, and some of the important points regarding this lesion have been apparently ignored or overlooked. I have found but occasional mention of primary miliary tuberculosis of the pleura in the text-books, monographs, and journal articles which I have consulted, and on the other hand, in several of the larger treatises on diseases of the pleura, Gerhardt's,<sup>1</sup> for instance, the disease is not mentioned at all. Still further, the real nature of these little nodules in the pulmonary pleura has sometimes been misinterpreted, for we find the statement "that miliary fibromata of the pleura is a common affection" (Schlodtmann)<sup>2</sup>. Weigert's<sup>3</sup> article, published in 1883, is the best which I have found, although he devotes less than half a page to the subject. He mentions, among other things, that when miliary tubercles of the pleura have existed for some time they can no longer be distinguished from miliary fibromata. The frequency of the lesion, the natural fate of the nodules, except in Weigert's brief statement just noted, and their relation to so-called primary tuberculous

<sup>1</sup> GERHARDT, C.: Diseases of the Pleura. *Deutsche Chirurgie*, 43 Lief.

<sup>2</sup> SCHLODTMANN: Zur patholog. Anatomie der Staubinhalationskrankheiten. *Centralbl. f. path. Anat.*, 1895, No. 16.

<sup>3</sup> WEIGERT, C.: Die Wege des Tuberkelgiftes zu den serösen Häuten. *Deutsche med. Wochensh.*, 1883, Nos. 31, 32.



pleurisy, have, so far as I know to the contrary, not been commented upon.

*Relative Frequency of Primary Pleural Tuberculosis.*

I have during the past three months examined the pleural surfaces of one hundred and thirty-one adults, which have come to autopsy. Children's lungs were not included in this list. The ages of the subjects varied between fourteen and ninety-two years. In thirty-seven cases, there was more or less advanced pulmonary tuberculosis. In three cases, the pleuræ on both sides were so covered with adhesions that the presence or absence of tubercles could not readily be determined. On gross inspection of the remaining ninety-one cases, in which the lungs were free from tuberculosis, in forty-five, or nearly fifty per cent., there were seen on the surface of the pulmonary pleura, and in one case on the costal pleura as well, certain nodules and patches which previous studies had led me to regard as being tuberculous in character. The gross diagnosis was confirmed by microscopic examination in all but four of these forty-five cases. In three of these cases the tiny raised nodules on the pleura proved to be miliary air cysts (see Fig. 4). In one case the nodules, six in number, were found to be miliary endotheliomata. In the forty-six remaining cases, the pleural surfaces as well as the parenchyma of the lung were free from visual evidences of tuberculosis. In several instances, however, sections were made of the pleura in these negative cases, and in three microscopic evidences of tubercle were found, *i. e.*, tuberculous foci too small to be determined by the unaided vision. The bronchial lymph nodes were found tuberculous fifteen times in the ninety-one cases, six times unassociated with tuberculosis of the lungs or pleura,



and nine times in connection with tuberculosis of the pleura alone.

*Channels of Infection in Miliary Tuberculosis in Cases in which the Lung is not Involved.*

The tubercle bacillus may become finally lodged in the sub-pleural connective tissue, either by means of the blood-vessels (hæmatogenous infection) or by the lymphatics (lymphogenous infection). In the case of the lymphatics, there are apparently six possible routes of infection: First, directly from the air vesicles of the lung through the lymphatics to the pleura; second, from the bronchial lymph nodes to the pleura; third, from the peritoneum; fourth, from the pericardium; fifth, from a tuberculous breast or rib adjacent; sixth, from tuberculous foci in the neck.

Notwithstanding that the current in the lymphatics of the lung is from the periphery toward the root, there is abundant evidence at hand, morphological as well as experimental, to show that particles of various kinds, bacteria, cellular elements, etc., may, when they obtain entrance into the blood circulation, or the lymph stream as well, travel in the reverse direction of the current almost as readily as with the flowing stream (rückläufige Transport). Fleiner<sup>1</sup> has shown, moreover, in a series of experiments, that various particles, especially red blood corpuscles, when allowed to escape into the air vesicles, were, within a few minutes, largely carried from thence through the lymphatics in both directions of the current, partly to the bronchial lymph nodes and partly to the pleura. Fleiner considers it very doubtful whether foreign particles of any sort deposited in the pleura ever find their way into the pleural sac, provided the surface endothelium or epithelium, as

<sup>1</sup> FLEINER: Ueber die Resorption corpuscul. Elemente durch Lungen und Pleura. *Virch. Arch.*, Bd. 112, 1888, S. 97.

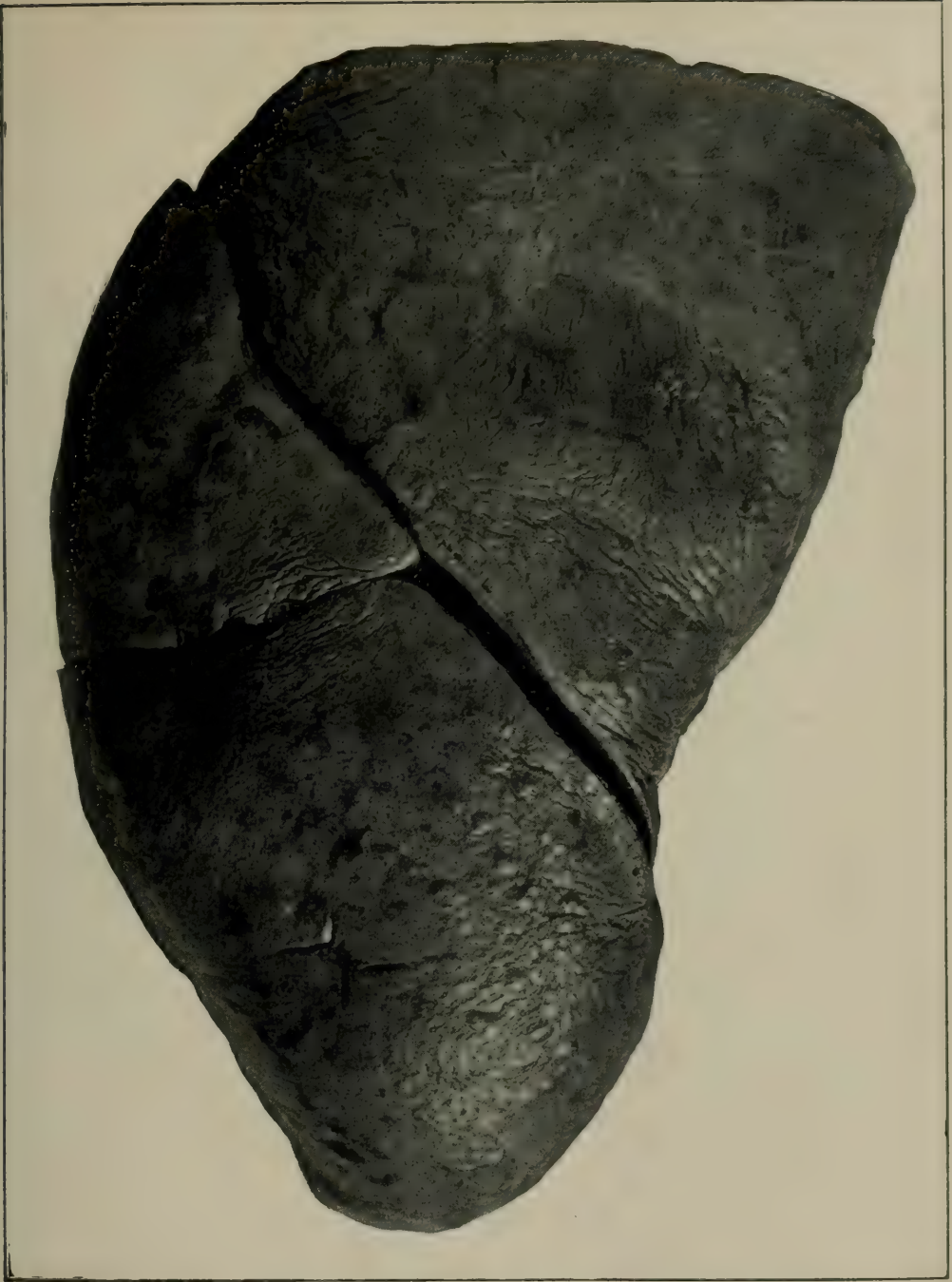


Fig. 1. Miliary Tubercles of the Pleura, Upper Lobe.









Fig. 2. Miliary Tubercles of the Pleura.





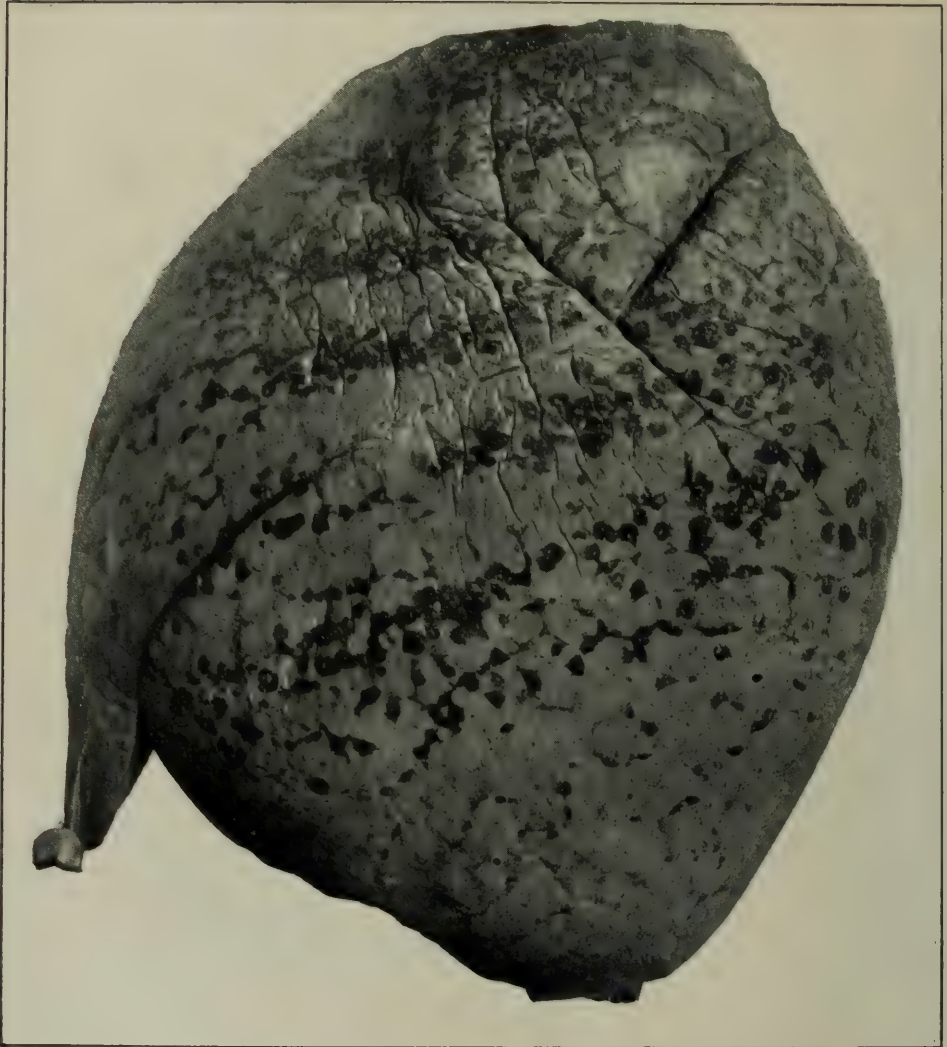


Fig. 3. Pleura Studded with Miliary Tubercles. A Tuberculous Patch near the Apex.

one chooses to call it, is intact. In one of my cases, for which I am indebted to Dr. F. C. Wood, the patient died of an acute tuberculous pleurisy, starting from an old tuberculous sinus in the neck. In this case both surfaces of the pleura on the affected side were thickly studded with fresh miliary tubercles. In the sac was a large amount of fresh exudate, serum, fibrin, and pus.

*Macroscopic Features of Miliary Tuberculosis of the Pleura.*

Miliary tubercles affecting the pleura vary somewhat in different cases. The pleuræ of one or both sides may be involved. In most cases the costal pleura is not involved. In number they vary from a single nodule to several hundred. No particular part of the pulmonary pleura seems especially apt to be invaded. As a rule, they are formed in the sub-pleural connective tissue, and are seen on the surface as flat or somewhat raised whitish structures, which vary in size from 1 to 5 mm. in diameter. Almost invariably they are surrounded by a distinct zone of pigment (coal-dust). Sometimes they are found surrounded by lymphoid tissue, Arnold's lymph nodules. Sometimes they are found embedded in the substance of the lymph nodes of the pleura first described by Heller.<sup>1</sup> On section, they frequently contain calcareous centres. In addition to these focal areas, there are not infrequently seen on the pleura more diffuse fibrous patches, which may be calcareous and which may show microscopically the other evidences of tuberculosis.

*Microscopic Features of Tubercles of the Pleura.*

The striking morphological peculiarity of tubercles of the pleura is the frequency with which the tendency

<sup>1</sup> HELLER: Ueber subpleurale Lymphdrusen. *Deutsches Arch. f. klin. Med.*, Bd. 55, pp. 141-145.

to fibrous-tissue metamorphosis is observed. Apparently the older the lesion the more dense and fibrous are the tubercles apt to become. In the substance of the nodules, but more especially surrounding them, a considerable accumulation of coal-dust in the form of tiny granules is invariably found. One is justified, it would seem, in assuming for the coal-dust deposited about the nodules an important rôle in the induction of the marked tendency to fibrous changes which they so commonly present. The many observations on this point, notably the now classical work of Julius Arnold<sup>1</sup> on Staub inhalation, prove conclusively that foreign inorganic particles lodged in the tissues are regularly followed by connective-tissue proliferation about them. Tubercles in the pleura are sometimes conglomerate. Giant cells, cheesy and calcareous degeneration, and other morphological characters of tuberculous inflammation are frequently met with. It was noted in several of my cases that different nodules from the same pleura evidently represented different stages of development. Some were so fibrous that they could not be differentiated from miliary fibromata; others contained cheesy centres, and still others giant and polyhedral-shaped cells in addition.

#### *Presence of Tubercle Bacilli.*

In but two cases were tubercle bacilli satisfactorily demonstrated in sections. Nor is this small number of cases in which the bacillus was demonstrated so surprising, when the fibrous nature of the nodules is taken into account. In chronic apex tuberculosis, the tubercle bacillus not infrequently disappears entirely from the

<sup>1</sup> ARNOLD, J.: Untersuchungen über Staubinhalation und Staubmetaste. Leipsic, 1885, F. C. W. Vogel.





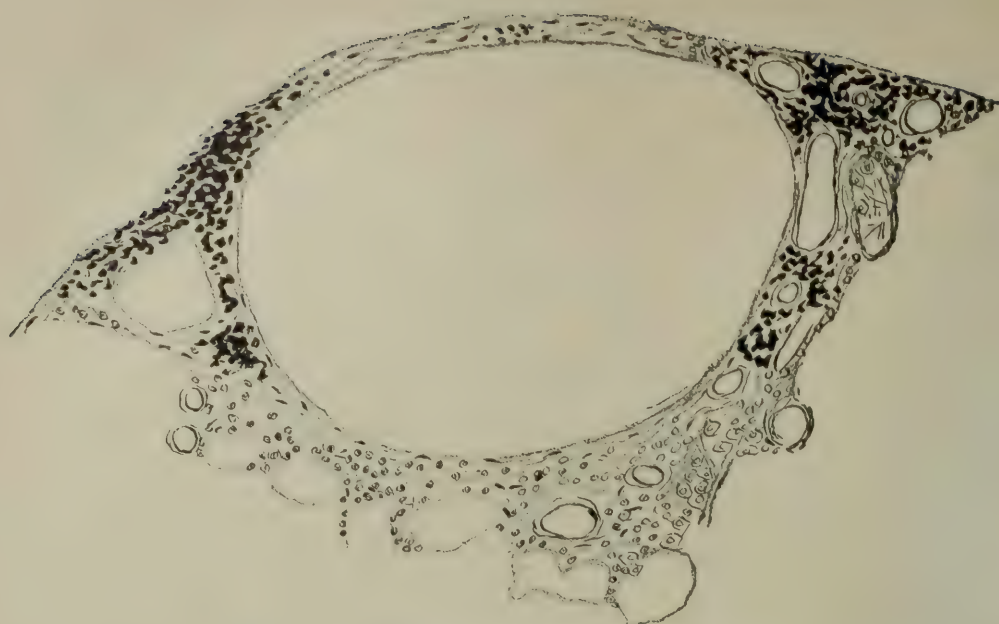


Fig. 4. A Miliary Air Cyst of the Pleura.

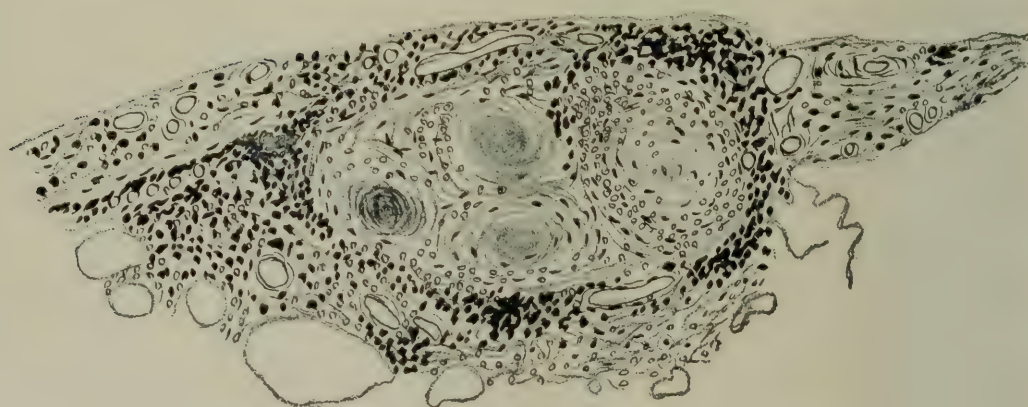


Fig. 5. A Conglomerate Tubercle of the Pleura.

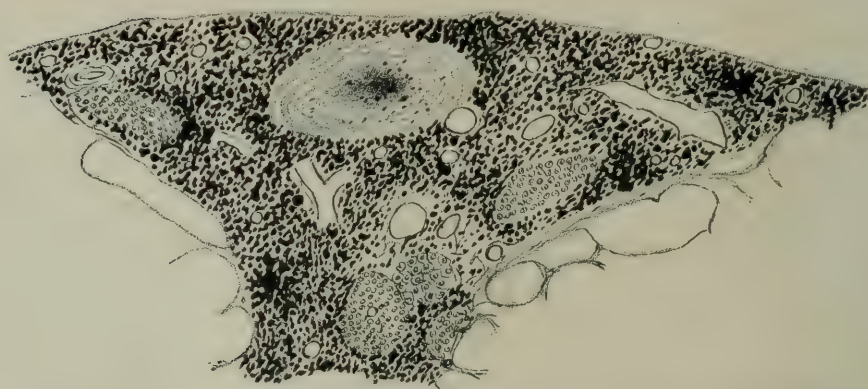


Fig. 6. A Miliary Tubercle of the Pleura, Surrounded with Coal-Dust Pigment.

lesion. This is notably also the case in chronic tubercles of the bronchial lymph nodes. As yet, inoculation experiments with tubercles from the pleura have not been undertaken by me.

While it is true that the lesions in general resulting from the growth of tubercle bacilli in the human body present no positive, characteristic morphological features, still it is also true that, in most cases, the appearances presented in microscopic section are so peculiar that it is but rarely that serious difficulty is encountered in diagnosis between miliary tubercles and nodules caused by other agents. In my cases, sections were made from different nodules in each case, and while in some of them, or in certain sections of the same nodule, the differential diagnosis between tubercle and fibroma was not clear, yet the examination of the cases, as a whole, revealed appearances which could fairly be regarded as being tuberculous in character.

### *Differential Diagnosis.*

Nodules which in gross appearance closely resemble miliary tubercles are sometimes observed in the pleura. These, however, can readily be distinguished in section from tubercles. Miliary air cysts of the subpleural connective tissue, or miliary lymphangiomata, are not uncommon. Miliary endotheliomata are also sometimes met with; and occasionally nodules which are apparently true fibromata are seen.

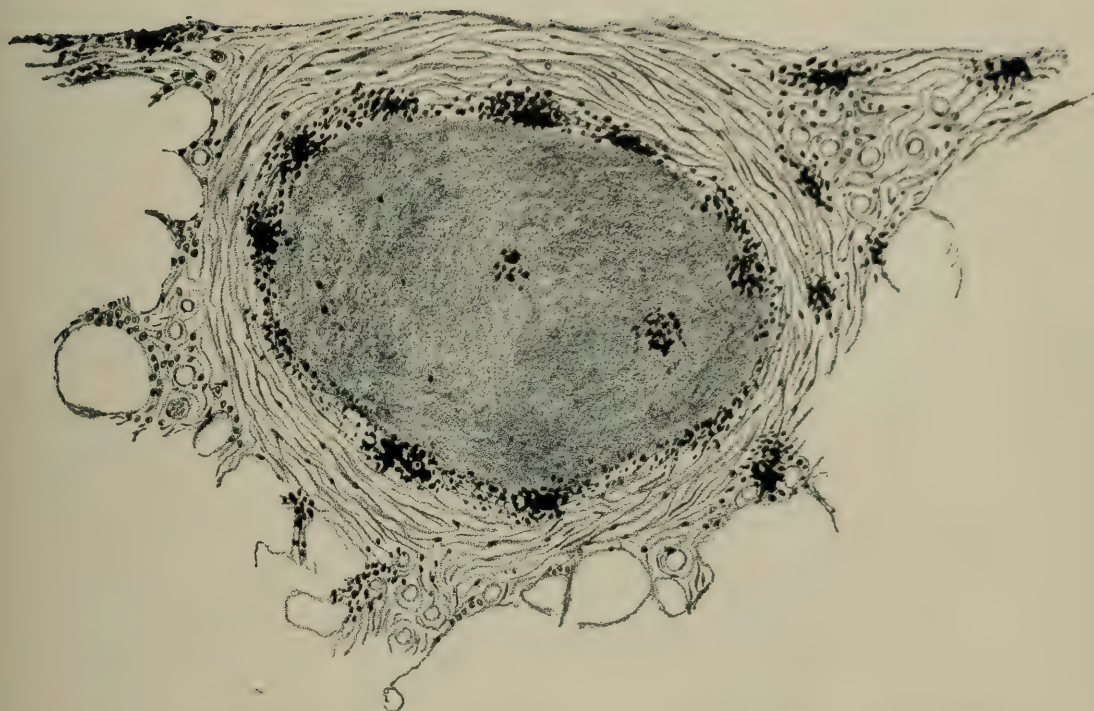
### *Relation between Miliary Tuberculosis of the Pleura and Acute Tuberculous Pleurisy.*

Much experimental evidence has accumulated of late years, showing that a large percentage of cases of acute pleurisy with effusion are really of tuberculous origin.



Much or most of this evidence has been based upon the result of animal inoculations of the serous exudate; and most of these published results have not been confirmed by post-mortem examination. The percentage of cases of acute tuberculous pleurisy, in which tubercle bacilli were found in the stained exudate, or in which positive evidence of tuberculosis was obtained by animal inoculation of the serous exudate, has thus far varied within wide limits in the published results of different observers, from ten per cent. to one hundred per cent. This wide range of results is, perhaps, partially accounted for, at least, by the selection, or want of selection, of cases adopted by different investigators, and especially by the different technique employed. Daminy inoculated large quantities of the serous exudate into susceptible animals, 300 c.c. in divided doses of 10 c.c., from each case, and claims positive results in all but two of more than fifty cases. The two negative results were in cases in which the quantity of serous effusion was scanty. In three cases of acute pleurisy with effusion, which have recently come to autopsy, I found in addition to the pleural exudate a crop of miliary tubercles embedded in the subpleural connective tissue. In one case (see Fig. 9) the tubercles were on the surface of the pleura and the cheesy areas communicated with the pleural sac. In these three cases the tubercle bacillus was not demonstrated in the exudate, nor was the serous fluid inoculated into animals. Were these cases examples of true tuberculous pleurisy, or were they examples of miliary tuberculosis of the pleura with concurrent or mixed infection?

It is difficult to decide in these cases, in the absence of important pathological data, but the presumption would seem to be strong that the miliary tubercles present were the determining factors of the new-formed



**Fig. 7.** A Miliary Tubercle of the Pleura, Surrounded by Fibrous Tissue and Composed Largely of Cheesy Material.







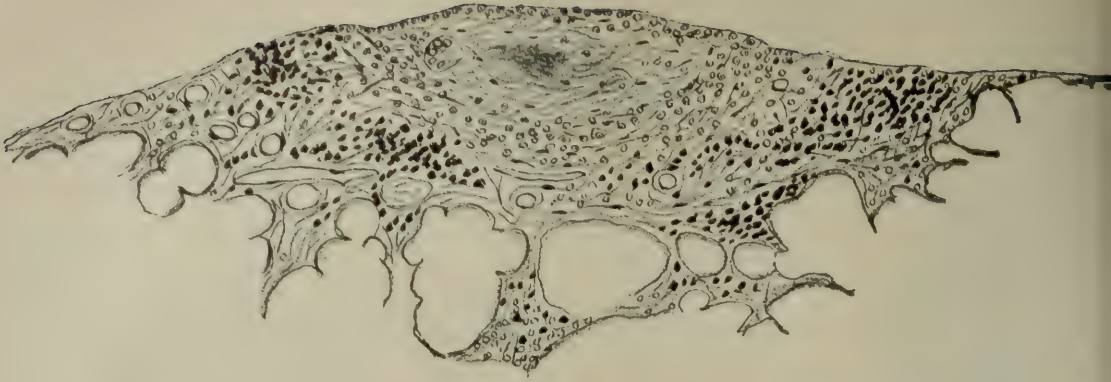


Fig. 8 A Miliary Tubercle of the Pleura, Containing a Giant Cell.

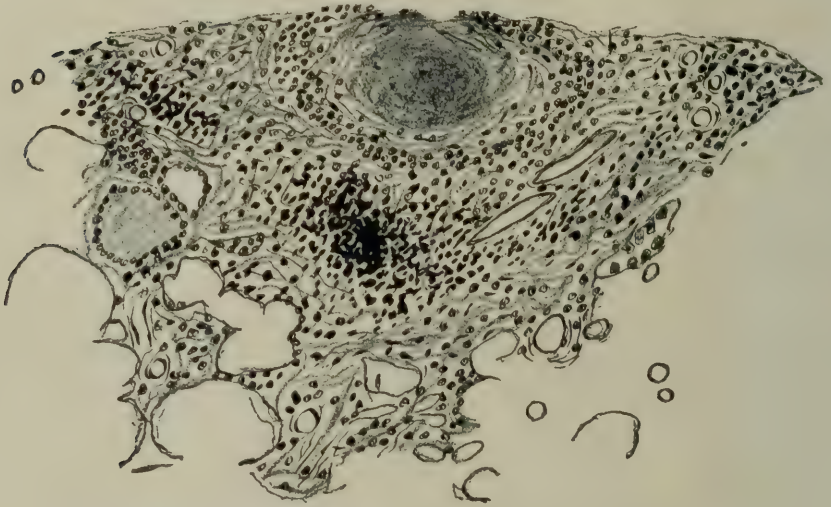


Fig. 9. A Cheesy Tubercle of the Pleura.

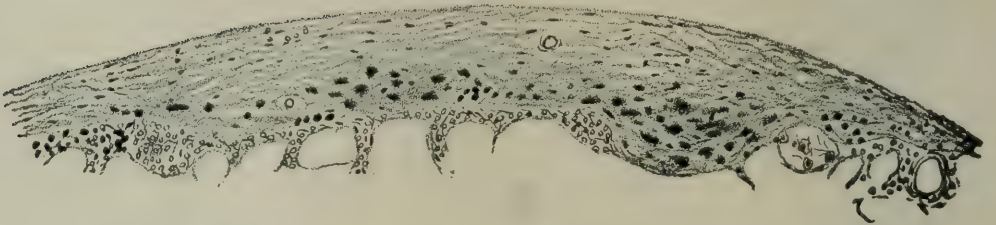


Fig. 10. Diffuse Tubercle Tissue of the Pleura, from Case of Multiple Miliary Tuberculosis of the Pleura.

exudate. This presumption is based upon: first, experimental observation, which indicates that it is very improbable that bacteria are ever capable of passing through the pleura into the pleural sac, provided its cellular lining be intact; second, experimental evidence that tubercle bacilli inoculated into the pleural cavities of susceptible animals are capable of inducing an acute exudative inflammation, with the formation of serum, fibrin, and pus; third, escape of tubercle bacilli into the pleural cavity is greatly facilitated in those cases of miliary tuberculosis of the pleura in which the tubercles involve the surface, and is almost inevitable when cheesy areas communicate with the sac.

It is not altogether improbable that pleural tubercles in susceptible individuals may occasionally serve as primary nidi for a subsequent tuberculous infection of the parenchyma of the lung.

#### *Conclusions.*

First: Miliary tuberculosis of the pleura, without other tuberculous manifestations of the lung, is of frequent occurrence.

Second: Miliary tubercles of the pleura may, apparently, assume unusual significance either in causing in susceptible individuals, or under otherwise favorable conditions, a generalized tuberculous exudative pleurisy; or by complicating, through concurrent infection, an acute exudative pleurisy of independent origin.

Third: Miliary tubercles in this situation are prone to become fibrous.

#### *Discussion.*

Dr. W. H. PARK said that at the health department laboratory guinea-pigs had been inoculated from a case of pleurisy with effusion, and tuberculosis had



resulted in these animals. The patient with pleurisy had, however, perfectly recovered after about two months. He would like to know whether tuberculin would bring out a reaction in such a case. If the affection was a latent tuberculosis this should occur, and under such circumstances it would be very puzzling to decide how much significance should be attached to the tuberculin test.

Dr. PRUDDEN said that while the work embodied in this paper did not absolutely exclude miliary fibromata, it made it clear that their occurrence was far less frequent than had been hitherto supposed.

---

*Stated Meeting May 10, 1899.*

T. MITCHELL PRUDDEN, M.D., PRESIDENT.

REPORT OF THE COMMITTEE ON MICROSCOPY ON THE  
CASE OF MIKE KELLY.

The report of the committee was read by Dr. HARLOW BROOKS. He stated that the committee had subjected to microscopical examination the tumors from the scalp and the larger tumors of the skin of other regions in the well-known case of Mike Kelly, of Bellevue Hospital. These growths had all shown substantially the same characteristics, viz., that of a highly cellular fibroma, somewhat resembling fibro-sarcoma, but having a blood supply which was hardly sufficient for a tumor of the latter class. In the small nodules from the small intestine the pigment had been found lying between the peritoneal and muscular coats, and not at all within the cells of the tumor growth proper. These tumors had been slightly different in some respects from those on the external surface of the body, but essentially the same in structure. They contained a good deal of involuntary muscle, appar-

ently derived from the coats of the intestines. These growths were made up of connective tissue containing a very large number of connective-tissue cells, some of these cells being apparently proliferating. He had not found any evidence of neuroma or of connection with nerve fibres. Sections of the tumor tissue were exhibited under the microscope.

#### A CASE OF ASEXUALISM.

The patient was twenty-nine years old, and died from the results of mitral stenosis without any exhibition of symptoms pointing to sexual organs. On removing the pelvic peritoneum a rudimentary testis was found on each side. No prostate gland could be made out. The penis appeared normal although undersized. There was nothing in any of the sections of the supposed genital glands which suggested either male or female genital gland. The appearance was more that of lymph gland. The scrotum contained no vestige of testicular tissue.

#### REPORT OF FIVE CASES OF SUDDEN DEATH FOLLOWING LODGMET OF FOREIGN BODIES IN THE LARYNX.

Dr. LEON T. LEWALD showed specimens from these cases. They had all occurred in males in middle or advanced life, and the foreign body had been in each case a piece of meat that had lodged in the pharynx just over the entrance to the larynx.

#### *Discussion.*

Dr. J. H. LARKIN said that about two years ago he had seen a man who, while eating soup, had swallowed a piece of meat-bone measuring about half an inch in diameter. It had lodged in the ventricle of the larynx. About three days later he entered the hospital much cyanosed and died one hour later. At the autopsy the

bone was discovered and around it a good deal of ulceration was noted. The cause of death had been œdema of the glottis.

Dr. NORTHROP remarked that he could recall a specimen presented some years ago to the Society by Dr. Hermann M. Biggs. It had been taken from a man who dropped dead instantaneously while eating, and without having any convulsions.

#### TUBERCULOUS MYOCARDITIS.

Dr. F. C. WOOD presented a specimen of tuberculous myocarditis removed from a child of nine months, at St. Luke's Hospital. At the autopsy no miliary tuberculosis was discovered. Firm white patches were found in the myocardium and on the surface of the ventricle. Only twenty-five or thirty cases of this form of tuberculosis were reported, although it was common enough in connection with miliary tuberculosis. The case reported in detail later.

#### TWO CASES OF AORTIC ANEURISM WITH RUPTURE INTO THE PULMONARY ARTERY.

Dr. LEWIS A. CONNER reported these cases, and presented the specimens. Both patients had come recently into the Hudson Street Hospital, and had died shortly after admission, so that the clinical records were incomplete. The first one, a man sixty-six years of age, a porter by occupation, was admitted on April 10th with feeble heart action and deep cyanosis, but with no subjective symptoms except weakness. He said that he had felt well up to three hours before, when he had begun to feel drowsy. He was almost pulseless, but was free from dyspnoea. Auscultation over the precordium had revealed nothing but extreme feebleness of the



heart sounds. The heart was much enlarged, and both ventricles were found at autopsy much dilated, and their walls moderately hypertrophied. The heart muscle showed slight fatty and granular degeneration. The valves were normal in appearance and seemed to be competent. The aorta from its origin to a point beyond the origin of the innominate artery was the seat of dilatation and many atheromatous areas. At a point 3 cm. above the aortic valve was a recent tear, 2 cm. long, opening into the left pulmonary artery. The lungs were much congested and somewhat œdematous.

The second case was that of a man of forty years, a Danish laborer, who was admitted on February 10th with a history of having been well up to twelve hours previously. Since that time he had been coughing up blood-stained sputum. While walking from the ambulance to the ward the pulse became weak, and he coughed up blood and serous fluid. He died in twenty minutes. At the autopsy the heart was found considerably enlarged, and the left ventricle much dilated, but its wall was of normal thickness. The wall of the right ventricle was greatly thickened. From the right sinus of Valsalva, immediately to the right of the orifice of the right coronary artery, was an aneurismal pouch the size of a walnut. It was filled with old clot, and seriously obstructed the flow of blood into the pulmonary artery. The aneurism had ruptured immediately behind the pulmonary valve. There was a clot extending into the pulmonary artery and almost occluding the right branch. The left lung was evidently œdematous, and the bronchi were filled with reddish fluid. In the upper part of the left upper lobe was an area of recent tuberculous consolidation, and there were evidences of an old tuberculosis. The aorta showed throughout considerable arterial sclerosis. The symp-

toms in these two cases presented a remarkable contrast, which it was not easy to explain by the morphological findings. In the first case it seemed as though the force of the current of blood in passing from the aneurism into the pulmonary artery must have been diminished; in the second case, the direction of the blood stream must have been upward into the pulmonary artery. He could not believe that the clot in the pulmonary artery had been there more than a very short time before death. Another interesting feature in the second case was the recent development of tuberculosis in a man of good physique. The association of this with a serious interference with the flow of blood into the lungs was exceedingly significant. Regarding the symptoms of rupture of aneurisms into the pulmonary artery, the speaker said that in nineteen reported cases there had been either a systolic bruit, a continuous murmur, or a double murmur corresponding to both systole and diastole. There had been in addition, the usual signs and symptoms of disturbance of the general circulation.

#### *Discussion.*

Dr. MARY PUTNAM-JACOBI suggested that the absence of the dyspnoea in one of the cases indicated that with the extreme cyanosis there had been more or less insensibility, and hence the lack of appreciation of the distress.

Dr. NORTHRUP said regarding the narrowing of the pulmonary artery and its relation to the development of tuberculosis, that he could recall six cases of congenital malformation in children, and one adult, in which there had been tuberculosis. It was well to remember that certain German surgeons claimed to treat tuberculosis of joints successfully by producing local cyanosis. He did not think the opinion was now so



generally held as formerly that tuberculosis was associated with a small pulmonary artery.

Dr. WOOD said that the surgeons attempted to produce local congestion in joint disease, and not a diminished blood supply, hoping thereby to control the tubercular process.

#### A CASE OF CANALIZED THROMBUS OF THE ILIAC ARTERY.

Dr. GEORGE P. BIGGS presented this specimen. It had been removed from a woman, thirty-seven years of age, who died from the result of mitral stenosis. The only history obtained was that she suffered from chronic valvular disease of the heart. The left common iliac artery was smaller than normal and very firm, as though filled with an organized thrombus. Transverse section of this thrombus showed ten longitudinal canals, each one-half to one millimetre in diameter, by means of which the circulation had been partially restored. The canals were chiefly in the periphery of the clot, and were found, on microscopical examination, to have an endothelial lining. The thrombus began 2 cm. from the aorta, and was 4.5 cm. in length, covering the opening of the internal iliac, and extending into the external iliac artery. No attempt had been made to follow the course of the collateral circulation, but it had been noted that the iliac vessels of the opposite side were unusually large.

#### A PIN IN THE VERMIFORM APPENDIX.

Dr. H. BROOKS exhibited this specimen, which had been taken from a child of five years. The illness had begun, six weeks before death, with convulsions, followed by pain in the right ear, stiffness of the neck, dilatation of the pupils, vomiting, restlessness, and slight fever. When admitted to the New York Hospital, there



had also been noted internal strabismus of the right eye. The patient had developed delirium, giving place to stupor several days before death. The temperature had been between  $99^{\circ}$  and  $100^{\circ}$  F. during the last two weeks of life, and the highest recorded  $101^{\circ}$  F. There had been no abdominal symptoms. The mother stated that eighteen months before death the child had swallowed a pin, which had never been found in the fæces. At the autopsy the dura mater had been tense, and the convolutions flattened. The dura was adherent to the right parietal lobe, and beneath the adhesions was a large single abscess, measuring 6.5 cm. vertically, 6.5 cm. antero-posteriorly, and 4 cm. transversely. The contents of the abscess consisted of 200 c.c. of thick, yellowish, offensive-smelling pus. The abscess occupied all the right parietal lobe except the ascending parietal convolution and the parietal portion of the convolution of the longitudinal fissure. There was destruction also of the posterior portion of the temporo-sphenoidal lobe. A fairly firm wall to the abscess had suggested that it might have existed for some time. Rupture into the subdural space was only prevented by the adhesions to the dura. The cerebral sinuses and both ears appeared normal. The vermiform appendix hung free in the peritoneal cavity, and measured 8 by .75 cm. There were adhesions of the omentum to the distal third of the appendix, opposite its mesenteric attachment. Slight traction on the omentum had caused a partial breaking of the adhesions, and had exposed an opening, 2 mm. in diameter, in the anterior wall of the appendix, 2.5 cm. from the tip. Through this opening had projected the head of an ordinary pin, 2.5 cm. in length, covered with rough concretions. The point of the pin, which was toward the cæcum, was also embedded in the anterior wall of the appendix, nearly causing a second perforation.

Dr. BROOKS said that he had reported some months ago a case of pin in the appendix. In that instance the head of the pin had perforated and formed a small localized abscess. There had been no symptoms referable to the appendix. He had learned that a series of thirty-three cases of this kind had been collected at the Johns Hopkins Hospital; that in the greater number of them the head of the pin had been found in the distal end of the appendix, and often there had been few direct symptoms referable to the appendix during life.

#### FISH-BONE IN THE ŒSOPHAGUS.

Dr. W. P. NORTHRUP exhibited a sharp triangular piece of fish-bone, having a sharp spur coming off from its base. He had removed it with the aid of laryngeal forceps after having first located it with his finger.

#### A CASE OF PURULENT PNEUMOCOCCUS MENINGITIS.

Dr. F. C. WOOD reported a case of meningitis in which the upper surface of the hemispheres had been covered with a thick layer of pus. The patient had been in the hospital for some weeks with a pneumonia of the left lower lobe. It was supposed that resolution had taken place, but the symptoms had suddenly become worse; then meningeal symptoms had supervened, and death had occurred shortly afterward. At the autopsy there had been a well-marked oedema and congestion of the left lower lobe, but no consolidation. The spleen had contained infarcts. In the heart was a small clot situated underneath one of the valves and attached to the myocardium. Cultures made from the blood during life had proved negative, but the pneumococcus had been obtained in smears made from the contents of the abscess.



## BILIARY CIRRHOSIS.

The second specimen presented by Dr. Wood had been taken from a well-nourished man who had been admitted in coma, with a subnormal temperature, and without any definite history. At the autopsy extreme jaundice had been noted, but no ascites. A number of small hemorrhages were found underneath the pericardium, and the stomach and duodenum contained a great deal of blood. The gall bladder was represented by a small mass of fibrous tissue without a lumen. In the common duct was a small oval transparent stone composed entirely of cholesterin. The surface of the left lobe of the liver was covered with small nodules. The case was evidently one of biliary cirrhosis. Microscopical examination of the liver had shown a thickening of the bile ducts and necrotic areas apparently the result of an old infection. There was also a diffuse intralobular cirrhosis. Some of the cells of the liver had been well preserved. The spleen was small.

*Discussion.*

Dr. BROOKS said that the first case recalled one that he had seen at the Harlem Hospital. There had been complete consolidation of the lower lobe of the lung at the time of admission. After about two weeks the patient developed a meningitis, apparently of the cerebrospinal type, and died. The autopsy had shown a resolving pneumonia on the left side and a thick greenish exudate over the convexity of the brain, extending down over the cord. Smears from the lung had exhibited only the diplococcus of Weichselbaum. He had not been able by cultures to find the pneumococcus in the lung, but it had been present in the trachea and bronchi. In the cerebral exudate the pneumococci and Weichselbaum



diplococci had been demonstrated. It was thought that the source of the trouble had been an infection of a bronchus with the Weichselbaum diplococcus, and that eventually it had been a case of mixed infection.

MALIGNANT ENDOCARDITIS—INFARCTS IN LUNG, SPLEEN,  
AND KIDNEYS.

Dr. L. A. CONNER referred to a case of malignant endocarditis which had resulted in death at the New York Hospital not long ago, with a very obscure history. A very large thrombus had been found filling up the whole tricuspid orifice. There had been not only many infarcts in the lungs, but one in the spleen and one in the left kidney as well. Nothing abnormal was found in the left side of the heart.

*Discussion.*

Dr. NORTHRUP remarked that in his experience the lesion had several times been in the right heart, and yet infarcts had been present in the spleen, lungs, and kidneys. It was his impression that this occurred frequently in primary ulcerative endocarditis.

OSTEOSARCOMA OF KNEE SIMULATING TUBERCULOUS  
JOINT DISEASE.

Dr. REGINALD H. SAYRE presented a tumor removed from a girl of thirteen years. According to the history, she had fallen last November, and struck the knee several times within a month. During the previous summer she had complained a little of pain in the knee. When first seen by him in the latter part of February, there had been some swelling of the upper part of the knee not unlike malignant disease. He had treated the case tentatively for a short time, thinking it possible that the

disease was tuberculosis. Within a few weeks the bone had increased rapidly in size, and on exploratory operation a large bleeding tumor had been found. He had then amputated at the hip joint. Microscopical examination had shown the tumor to be a large spindle-cell sarcoma. The child had improved rapidly after the amputation, and had been discharged within two weeks.

In this connection Dr. Sayre exhibited a skiagraph of a round-cell sarcoma of the tibia that he had presented to this Society about one year ago; also a skiagraph of a case in which there had been doubt as to whether the diseased condition of the fibula had been syphilitic or malignant. The skiagraph had proved disappointing as a means of differentiating between syphilitic and malignant disease of bone.

#### *Discussion.*

Dr. JAMES EWING remarked that skiagraphy should be of service in differentiating between round-cell and spindle-cell sarcoma of bone, for in the former, the shaft of the bone was usually destroyed very early.

---

*Stated Meeting, October 11, 1899.*

T. MITCHELL PRUDDEN, M.D., PRESIDENT.

CARCINOMA GELATINOSUM MAMMÆ (CARCINOMA MUCOSUM).

Dr. E. HODENPYL showed a specimen of this kind. He said that it was of comparatively rare occurrence; and when the specimen first came into his possession he imagined it might be almost unique, at least so far as the situation of the lesion was concerned. He had found, however, in Ziegler's *Pathology* a very good short descrip-

tion of gelatinous carcinomata of the breast, with a drawing of a case similar to his own, and in Bruns's *Beiträge*, 1896, vol. xvi., he had found an elaborate monograph on this subject by Dr. Fritz Lange. The latter contained a *résumé* of all the recorded cases, seventy-five in number. Roger Williams, in his new text-book on *Tumors of the Breast*, stated that, although the disease had been described, he had never met with a case. The patient from which this tumor had been removed was admitted into Roosevelt Hospital, on account of a tumor in the left breast. She stated that it had been there "as far back as she could remember." For many years it had been about the size of an egg; was hard and painless, and showed little if any tendency to increase in size. A year and a half before its removal the mass had been struck a severe blow, in a fall, and after this it increased in size. About three months before coming under observation she accidentally struck the breast again. Shortly after this an opening formed near the nipple, from which dark bloody fluid escaped. Since this second traumatism the increase in growth had been quite perceptible, and the breast had become tender. Examination had shown a tumor, about four inches in diameter, which was unusually hard. The mass was freely movable, and apparently not adherent to the pectoral muscle. No axillary lymph nodes were palpable; there were no disturbances of sensation in the left arm; there was no cancerous cachexia. The patient stated that she had recently lost some flesh and strength. When Dr. McBurney first saw the case, he was in some doubt as to its nature, and had accordingly requested the speaker to make a preliminary microscopical examination at the time of operation. The examination was, in this case, particularly puzzling, for the reason that the mass submitted and removed from the densest portion of



the tumor resembled a quantity of jelly-like substance. On section, it presented a general alveolar arrangement of the connective tissue, the spaces of which were filled with a homogeneous, transparent, jelly-like substance. He concluded that the tumor was a carcinoma which had undergone extensive degeneration. The entire breast, including the axillary contents and pectoral muscle, was removed, and the patient made a satisfactory recovery. The tumor, which was exhibited, was hardened in formalin. It had to a great extent replaced the mammary gland and consisted largely of a soft, jelly-like substance. In places there were old hemorrhagic extravasations. It was partially encapsulated and contained some calcareous patches. There was a single enlarged lymph node between the tumor and the pectoral muscle, and two or three very small axillary nodes had been dissected out. In the microscopical examination made by Dr. Hodenpyl he had found nothing in the central portion other than alveolar arrangement, the spaces being filled up with a gelatinous material. It was only near the periphery that one could find the usual evidences of carcinoma—alveoli filled with epithelial cells. The lymph nodes were not involved. In all of the sections of the tumor, as well as the lymph nodes at a distance from the tumor, the blood-vessels contained numerous rather large and intensely black granules. Some of these granules were also scattered about in the tissues. The nature of these granules of pigment was afterward explained, when it was learned that the wound made in removing the specimen for microscopic diagnosis had been treated with the actual cautery to control hemorrhage. Dr. Hodenpyl said that, according to Lange, this gelatinous type was very much less malignant than the ordinary form of cancer of the breast. In many of the reported cases the tumor had existed for from six to eight years, and, in one instance,

for twenty-three years before removal. As a rule, the tumors were not larger than a walnut. Occasionally they reached the size of an egg. Ordinarily there was no lymph-node enlargement. Calcareous degeneration was quite a common feature. Metastases had not been very common. Out of twelve recorded autopsies of individuals who died after removal of gelatinous cancers of the breast, metastases had been found in only five cases, and these had been most often present in the lungs or in the liver. Local recurrence had not been a frequent feature. Three of the cases had been peculiarly interesting, in that the individuals had carcinoma of both breasts. In one case the carcinoma on one side was gelatinous, and on the other side it was of the ordinary scirrhus variety. In another case the gelatinous form had complicated a Paget's carcinoma of the opposite breast. In still another, the carcinoma had been gelatinous on both sides. One instance had been reported in which it had occurred in the breast of a male. Lange had thrown no additional light upon the chemical nature of this peculiar substance. Johannes Müller had examined chemically a number of these cases without finding any mucin in them, but other observers were quoted who claimed to have found mucin. With regard to the source of origin of the gelatinous substance, Lange, agreeing with other observers, believed that, unlike the ordinary form of so-called "colloid carcinoma" of the stomach, it did not originate within the epithelial cells of the tumor. The jelly-like substance, as it increased in amount, caused a secondary atrophy and more or less total obliteration of the epithelium. This statement seemed to be borne out by sections from the speaker's case. It appeared probable that the gelatinous material had its origin in the connective-tissue elements of the growth, but more investigation was needed to clear up this point.



CARCINOMA GELATINOSUM OF THE STOMACH—CARCINOMA  
GELATINOSUM OF THE LUNG.

Dr. J. H. LARKIN presented these two specimens of carcinoma gelatinosum in connection with the preceding. The first specimen was one of carcinoma gelatinosum of the stomach which had been removed from a man fifty-four years of age, who had given a history pointing to carcinoma, dating back six or seven months. The tumor in question occupied the anterior and posterior walls of the stomach, extending from the cardia to the pyloric end, along the lesser curvature, the only portion of the stomach not involved being the greater curvature at the pendent portion. The thickness of the stomach wall involved by carcinoma was about one to one and a half inches, the portion not involved being greatly thinned out. The pylorus was almost entirely occluded. The inner wall of the stomach over the infiltrating mass was changed into a trembling jelly. The peritoneum over the greater curvature was glistening and shining, while that over the tumor was lost and studded with myriads of tiny gelatinous masses. These small jelly-like bodies were also present on the mesentery and the under surface of the diaphragm. There were no metastases in the other viscera. The noteworthy features of this specimen were the abrupt stoppage between the carcinoma and the more normal stomach wall at the greater curvature, the great thickness of the stomach wall and the direct extension of the infiltration through it, and the appearance of myriads of gelatinous masses in the mesentery.

Dr. LARKIN also exhibited a

## GELATINOUS CARCINOMA OF THE PERITONEUM.

The tumor originated primarily in the peritoneum. It



had been taken from a woman, aged sixty-five years, who had not given any history of previous illness. She had died of chronic Bright's disease. At the autopsy myriads of small gelatinous masses under the pleura and in the substance of the lung were discovered. A thorough search had been made in the peritoneum, and alongside of the coeliac axis a small mass, about three-quarters of an inch in diameter, had been found. This had been removed, and it had then been found that the mesenteric glands back of the mass were enlarged and gelatinous. On microscopical examination of the tumor in the peritoneum it was found to be made up of irregular connective-tissue spaces lined with high cylindrical epithelium. The cavity was filled with gelatinous material, staining with hæmatoxylin, containing a few cells. The same appearance was presented in a node found on the superior surface of the liver. The small gelatinous metastases in the lung had presented the same irregular network of connective tissue made up of the connective tissue of the old walls of the air vesicles. The characteristics of the other tumors were still preserved. In the masses in the lung the mucous material was very abundant. The speaker asked for information regarding the proper classification of tumors of this kind.

#### *Discussion.*

Dr. P. A. LEVENE said that even mucin had not been studied very much by the physiological chemists. It had been considered to be a proteid combined with sugar. If considered to be a glucoside, one could not explain its pathological origin as a product of decomposition. If mucin was a proteid molecule having in it a carbohydrate, one would be inclined at the present day to look upon this material as a product of the function of the cell rather than of its decomposition. He was at present

working on one of the mucins, and as far as he had gone he had been led to look upon it as a product of decomposition. He thought mucin consisted of two parts, one part being the product of metabolism—an acid which subsequently combined with a proteid.

Dr. ERNEST E. SMITH thought a good deal of confusion existed among chemists as well as among pathologists as to what mucin was, because they had attempted to include in the class of mucins all mucilaginous substances in the body. At one time he had undertaken a study of the mucous material of the alimentary tract, and had found that it was not mucin. He did not believe that the slimy material in the bladder was mucin at all. The mucous material of the intestine did not conform to the rather dogmatic requirements of chemists for this class of bodies, viz., that it should be composed of carbon, hydrogen, sulphur, nitrogen, and oxygen, and that it should give a reducing substance when boiled with acids. Of course, true mucin was found in saliva and human bile. The other class of mucilaginous substances seemed to be more closely allied to the nucleoproteids. Bladder and intestinal mucus, in his opinion, were of this latter class. He thought it probable that the gelatinous substances which were the result of decomposition or degeneration were allied to this class rather than to true mucin.

Dr. LEVENE said that Swedish investigators had shown that the mucin of the bile was not a true mucin, but a nucleoproteid. The trouble had been that formerly every proteid that gave a reduction test was considered a mucin, but it was now known that all the nucleoproteids contained a carbohydrate molecule. In his opinion, all the nuclein forms could be distinguished from mucin histologically by their behavior with staining agents.

Dr. SMITH added that since the first statement had been made, that the mucous substance of the bile was



a nucleoproteid and not mucin, the substance occurring in human bile had been re-investigated, and it had been found that mucin did occur in human bile. The original substance investigated had been ox bile, and this did contain nucleoproteid, and not mucin. The fact that nucleoproteids yielded a reducing substance when boiled with acid was no reason for confusing these compounds with true mucins, since, as already said, mucins contained only carbon, hydrogen, sulphur, nitrogen, and oxygen, while the nucleoproteids contained phosphorus in addition to these.

Dr. PRUDDEN said that he did not know why in this country we were calling these tumors "colloid," as they did not contain the substance giving the chemical reactions of colloid as it was found in the thyroid gland. Germans called them "gelatinous," and this seemed to be a more appropriate term. There was a large group of hyaline materials appearing under a variety of pathological conditions in the body, and which, with our present knowledge, could not be definitely separated one from the other. It was perhaps wise, under these circumstances, to consider them as a group of hyaline substances which were yet to be differentiated by the chemists, and to which we should attach special names as the result of superficial micro-chemical tests only with considerable reserve. The metastases in the lung of Dr. Larkin's gelatinous carcinoma seemed especially interesting because in places, instead of forming its own stroma as it grew, it had made use of the walls of the air vesicles, so that in places these vesicular walls, recognizable by their capillaries, by pigment, and by form, were to be seen lined with cuboidal and cylindrical epithelium and filled with gelatinous substance. Under ordinary circumstances in the growth of a carcinoma, the increase in stroma and in epithelium went hand-in-hand,



and one might well be in doubt which took the initiative. But here, the epithelial cells, having an appropriate base, seemed to have gone ahead on their own account. Thus these metastases, in addition to their intrinsic importance, seemed to afford a half-glimpse into that border-land of cell impulse toward which attention was so generally turning to-day.

#### TUMOR OF THE NYMPHÆ.

This specimen was exhibited by Dr. LARKIN for Dr. MATHEWS. It was a large tumor that had been removed from the nymphæ of a negress, twenty-four years of age, who had given a definite history of syphilis, and of this growth having existed for one year. Microscopically the tumor was made up of œdematous connective-tissue containing numerous blood-vessels surrounded, for the most part, by small lymphoid cells. The question arose as to whether syphilis had anything to do with the tumor. Personally he did not think it had.

#### TWO CASES OF ABSENCE OF ONE KIDNEY ASSOCIATED WITH PRESENCE OF A DOUBLE UTERUS.

Dr. LEON T. LEWALD presented these cases. He said that while he had met with these two cases within a few days of each other, a search of the literature had confirmed his former opinion that they must be extremely rare. The left kidney had been **absent in both instances**. The first patient, a woman of forty-seven years, had died of an acute lobar pneumonia. There was only one ureteral opening in the bladder, and no trace of a ureter or kidney could be found on the left side. The kidney was double the normal size and weight. The internal genital organs showed a malformation in the shape of a distinctly double uterus of the type known as "uterus

bicornis." In the second case, that of a woman aged thirty-two years, there had been a pyonephrosis and a perinephritic abscess. An incision had been made into the abscess and into the substance of the organ. So far as known, no cystoscopic examination had been made, hence if the operation had been pushed further the only kidney present might easily have been extirpated. In this case there was also present a double uterus, but of a different type from the first case. Externally the uterus departed from the normal only in the appearance of a slight depression at the middle of the fundus, but internally it presented two distinct cavities opening into a single cervix—"uterus bilocularis." The absence of the left kidney was believed to be more common than that of the right. The relation between this anomaly and the occurrence of double uterus had not been determined.

#### *Discussion.*

Dr. LARKIN said that he had met with two cases of absence of the kidney—in one it was the left, and in the other the right kidney that was missing. In one of these cases there had also been an adenoma of the kidney.

#### DIVERTICULUM OF THE BLADDER.

Dr. A. V. MOSCHOWITZ presented this specimen that had been taken from a man seventy-three years of age, who had been admitted to the surgical ward of the Mount Sinai Hospital with the presumptive diagnosis of specific urethritis. The speaker had examined the patient at the time of his admission, and had found the body temperature to be 103.4° F. and the pulse 14c. The patient stated that he had noted no special difficulty with urination up to one week previously, at which time there had been acute retention. Repeated but nearly futile

attempts at catheterization had been made before coming to the hospital. The skin covering the dorsum of the glans penis was black and necrotic; there was a fetid purulent discharge from the urethra. There was a hard swelling a little back of the meatus. A diagnosis of impacted calculus in the urethra had been made, and after irrigation a calculus had been easily removed with the aid of forceps. A catheter of large size, and even a Thompson searcher, could then be introduced into the bladder without difficulty. The urethra and the bladder were freely irrigated, but there was nevertheless a continuous discharge of blood and pus, and the patient was badly septic. One week later a suprapubic cystotomy was accordingly performed, and with the patient in the Trendelenburg position three calculi were removed. He had been surprised to note that at each inspiration, instead of at much longer intervals, there had been a jet of urine from one of the ureters. Closer inspection showed the opening to be not the ureter, but a diverticulum of the bladder filled with stones; these were removed. The patient had died of septicæmia the following day. Only a partial autopsy had been permitted. One ureter, instead of being in the natural place, was situated on the anterior portion of a diverticulum of the bladder. There were forty calculi in the diverticulum and three in the bladder proper.

Dr. MOSCHOWITZ further presented specimens from a case of

#### TUBERCULOSIS OF THE GENITAL TRACT.

They had been taken from a man who had been admitted to the Mount Sinai Hospital with a swelling of the left epididymis. Nothing abnormal was felt in the spermatic cord or vas deferens. On rectal examination the corresponding seminal vesicle presented a nipple-like



projection into the rectum. The case was considered an ideal one for a radical operation. Accordingly, an incision had been made along the scrotum and the testicle everted. The entire epididymis had then been dissected off without injury to the testicle. While the spermatic artery was compressed the testicle was inspected. As much of the vas deferens as possible was pulled out and a catgut ligature applied to the stump as a guide; then, the patient having been placed in the lithotomy position, a curved incision had been made around the anus, extending from one tuberosity to the other, and then by blunt dissection he had been able to reach up to and beyond the prostate. The seminal vesicle was then extirpated, being cut off as closely as possible to the ejaculatory duct. He had then taken hold of the stump and withdrawn the remainder of the vas deferens. It was now two months since the operation, and there had been no complication and no evidence of tuberculosis in the genital tract. The enlarged epididymis and the other organs, examined by Dr. Mandlebaum, pathologist to the hospital, showed numerous tuberculous abscesses and cheesy deposits. There was pus throughout the vas deferens and in the seminal vesicle.

#### CHANGED APPEARANCE OF DIPHTHERIA BACILLUS.

Dr. W. H. PARK said that in having a photograph made recently of a diphtheria bacillus that had been under constant cultivation for four years, he had been struck with the change in its appearance. It was now twice as long as before and more slender, and did not present the appearance of the diphtheria bacillus, while before it was very characteristic in its clubbed forms.

#### ANTITOXIC GLOBULINS.

Dr. PARK, on behalf of Mr. JAMES P. ATKINSON, the

assistant chemist in the research laboratory of the New York Health Department, presented a communication on the subject of antitoxic globulins. He stated that for two years past Mr. Atkinson had been testing the globulin in the antitoxin horses. He had thus found that the globulins increased as the antitoxin in the blood increased. It had been found that the globulin of normal horse serum and diphtheritic antitoxic horse serum, after separation from the serum by means of magnesium sulphate, might be separated into five fractions by the following method: The watery globulin solution was saturated with sodium chloride. This yielded a precipitate at the normal temperature. After filtering off this precipitate, a little more sodium chloride was added to the filtrate to insure saturation, and the temperature raised to  $40^{\circ}$  C. A turbidity appeared which, on raising the temperature higher, separated out in flocculi. At  $45^{\circ}$  C. the precipitate could be filtered off. At  $49^{\circ}$  C. the third precipitate commenced to come down, and at  $53^{\circ}$  C. it might be filtered off. The fourth precipitate came down between  $57^{\circ}$  and  $62^{\circ}$  C., and the fifth precipitate came out between  $67^{\circ}$  and  $72^{\circ}$  C. These precipitates were almost completely soluble in water. The  $67-72^{\circ}$  precipitate left a slight residue in water, but was soluble in dilute soda solution. An interesting feature of this process was that each one of these precipitates was antitoxic, and that the total globulin of the blood appeared to contain all the antitoxin, the moderate amount lost in the final precipitates being due, it is believed, to the effect of the heat used. Dr. Park added that they were going to give the antitoxic globulins to children instead of the antitoxic horse serum, to see if the rashes could not in that way be eliminated. By experimenting with these five globulins it was thought not improbable that more light could be obtained on this subject.

*Discussion.*

Dr. P. A. LEVENE said that it would be interesting to know if these different globulin precipitates varied in their physical characteristics. He was reminded of the experiments made at one time on Koch's tuberculin by Professor Kuhne. The conclusion had been reached in these experiments, that the toxin of the tuberculin was not chemically combined with these albumoses, but was merely carried down mechanically. The more flocculent the precipitate the more likely was it to carry down the antitoxin mechanically. As to the increase of globulins in antitoxic horses he was not sure that the increase of the globulin was due entirely to the abnormal condition of the horse. In a study of artificial diabetes that he had made he had found that the quantity of globulin had increased as a result of the pathological state which existed.

Dr. PARK replied that the horses which yielded the most antitoxin and seemed to be in the best condition yielded the most globulin. So far as he had observed, all these five globulins looked alike. As long as there was some globulin left in the serum from immunized horses, there was some antitoxin. No globulin from antitoxin serum could be extracted that was not, at least, feebly antitoxic.

---

*Stated Meeting, November 8, 1899.*

T. MITCHELL PRUDDEN, M.D., PRESIDENT.

A CASE OF HEMORRHAGIC INFARCTION OF THE SMALL  
INTESTINE.

Dr. LEON T. LEWALD presented this specimen. At the autopsy a thrombus had been found in the superior



mesenteric artery. It had evidently caused a hemorrhagic infarction and necrosis of the intestine, followed by perforation, general peritonitis, and death. The patient had been admitted to the hospital about twelve days before death, so that the infarction had evidently occurred while in the hospital. It had not been diagnosed during life. The man had been a heavy drinker. He had been well and working up to three months previous to coming to hospital. He had then begun to suffer from shortness of breath on exertion. For the past two months there had been increasing shortness of breath with attacks of orthopnoea and moderate whitish expectoration—never bloody. About three weeks before admission his feet had become swollen, and had remained so since that time. The urine had diminished in quantity. He was poorly nourished, and the skin presented a yellowish appearance. The chest was barrel-shaped, and the percussion note was of a wooden quality, the expiration being prolonged and of low pitch. The cardiac dulness was indistinct. No murmurs could be heard. The pulse was rapid, small, and of high tension, and there was considerable thickening of the arteries. The liver extended just below the free border of the ribs. The extremities were slightly œdematous. Two days later, or on February 25th, it had been noted that there was less dyspnoea and a better pulse. He had been treated for chronic nephritis. On February 26th he had expectorated some bloody fluid, and yet two days later he had been much better. Over the sternum a soft, blowing diastolic murmur was heard at this time, and the pulse was large, full, quick, and soft. On March 8th it was noted that the patient had been markedly worse for three days, and had complained of cramp-like pain in the epigastrium. Over the base of the right lung were heard numerous crepitant râles; over the left lung,

increased dulness. A soft, blowing diastolic murmur continued to be heard over the sternum. The pulse was small and wiry. He was vomiting frequently. On admission his temperature had been 99.3° F. On March 5th, or at the time when he had complained of the epigastric pain, his temperature had been normal. Two days later it had risen to 103°. On March 9th he had died. At the autopsy, the lungs had been found congested, and at the base of the right lung was a large hemorrhagic infarction. The heart was enlarged and dilated. There was extensive atheroma of the aorta, with calcification. The kidneys were enlarged, each weighing eight ounces. The capsules were adherent, and their surfaces were granular. There was an infarction in the right kidney. The liver weighed only three pounds ten ounces. Its surface was smooth. On section it was of a pale, yellowish color, and the lobules were indistinct. The spleen was small, weighing four ounces, and on section showed an increase in connective tissue. The intestines were matted together by fibrinous adhesions, and there was a large mass of intestinal coils in the umbilical region. On separating these adhesions a perforation had been found in a coil of the ileum, about three feet above the junction of the ileum with the cæcum. The intestine for a distance of five inches on either side of the perforation was dark and gangrenous, and on opening the bowel the mucous membrane in this region was softened, and the walls were dark, and infiltrated with blood. The corresponding mesentery was in a similar condition. On opening the superior mesenteric artery a firm thrombus had been found in one of the branches of the artery corresponding to the blood supply of the necrosed part of the intestine. The speaker said that the fact that there were infarctions of the lung, kidney, and intestine would point to an embolic process



followed by thrombosis. Fatal cases of this kind had not been very numerous. In one reported case in the *Glasgow Journal* the symptoms had been very similar—shortness of breath, dropsy of the extremities, and a few small hemorrhages from the lung, and severe colicky pains in the abdomen, with bloody vomiting a few days before death. This case had also presented a hemorrhagic infarction of the intestine, although there was no perforation. The clinical diagnosis was difficult, but the sharp pain in the abdomen and the absence of the usual signs of strangulation were significant.

*Discussion:*

Dr. E. HODENPYL referred to a case of thrombosis of the superior mesenteric vein recently seen at autopsy, in which the symptoms had been quite different. The patient, a man aged thirty-five years, had been seized four days before death with rather severe colicky pain in the abdomen. There had been no disorder of the bowel. The man had not seemed to be very sick at first, but had come to the hospital in an ambulance from a long distance, and on reaching the hospital had been in collapse. The autopsy showed a total thrombosis of the superior mesenteric vein, including all of its branches. There was a little atheroma of the aorta, but no history of syphilis could be obtained. The mesentery itself had been oedematous. With the exception of a moderate amount of phlebitis of the vein there was no clue to the origin of the process. There had been no elevation of temperature.

REPORT OF A CHEMICAL EXAMINATION OF A KNIFE-GRINDER'S LUNG.

Dr. EUGENE HODENPYL said that the subject of this



report was a knife-grinder, thirty-five years of age, who had died of pernicious anæmia. The history was that, he had worked at his trade for fifteen years. For the first ten years, he was employed as a grinder and worked in a large room with some forty others; for the past five years he had worked in a very small and illy ventilated room at the same occupation with some seven others similarly employed. The lungs presented a maximum degree of pigmentation, and it had, therefore, occurred to the speaker that it might be instructive to determine the amount of carbon contained in the lungs, and, if possible, the amount of emery and iron also. Such an investigation seemed especially desirable, since the speaker had been at the time studying the literature of "Staubinhalation" without finding a single case in which the amount of carbon had been determined in similar cases of anthracosis, and, moreover, upon inquiring among his colleagues, he had found that none had the slightest idea as to the amount of carbon which might reasonably be expected to be obtained from such a lung. There were many reports in literature, notably those by Arnold, in which gold and silver had been extracted from the lungs of artisans working with these metals, but no case had been observed in which the amount of carbon, emery, and iron had been determined in the lungs of knife-grinders. The technique employed was to digest the lung, which weighed 900 gm., and then obtain the charcoal, emery, and iron by precipitation. The lung was cut into small pieces, placed in a little water, to which were added two ounces of Johnson's preparation of papoid and enough hydrochloric acid to give a reaction of free acid in the solution. This mixture was kept at a temperature of 40° C. for ten days, when the lung became completely fluidified. It was then necessary, on account of the viscidness of the mass, to add

large quantities of water, in order to secure precipitation. About sixty gallons of water were added, and this mixture was allowed to stand in a number of tall jars for many days until precipitation was complete. The precipitate was then repeatedly washed in water until it was believed that all of the substance soluble in water had been removed. It was then evaporated to dryness and powdered. At this juncture, Mr. Allan C. Eustis and Mr. A. N. Richards, assistants in the department of physiological chemistry of the College of Physicians and Surgeons, New York, kindly undertook the chemical examination, and the speaker took this opportunity of extending his thanks to these gentlemen for the very complete analysis which they had made.

Analysis of lung taken from the body of a knife-grinder :

Total weight of lung dried and powdered, 48.1009 gm. Total solids, 44.7986 gm.; water, 3.3023 gm.

Soluble in ether, 14.6017 gm.; insoluble in ether, 30.1969 gm.

Composition of the portion which was soluble in ether: Free fatty acids, 7.498 gm.; neutral fats, 4.044 gm.; cholesterin, 3.037 gm.; (lecithins?)

Composition of portion insoluble in ether: Proteids, melanins, etc. (total nitrogen  $\times$  6.25), 15.4759 gm.; charcoal (total carbon—proteid carbon), 7.1989 gm.; ash, 4.29095 gm.

Composition of ash:  $K_2O$ , 0.2167 gm.;  $Na_2O$ , 0.3523 gm.;  $CaO$ , 0.0965 gm.;  $Fe_2O_3$ , 0.0879 gm.;  $Al_2O_3$ , 1.4628 gm.;  $SO_3$ , 0.0704 gm.;  $P_2O_5$ , 0.9565 gm.;  $SiO_2$ , 1.20434 gm.

Dr. HODENPYL said that, on first receiving this report, he had been somewhat disappointed that the amount of carbon was not greater, but since then he had made some simple experiments which demonstrated that,

after all, 7 gm. + of this particular charcoal was really an enormous amount to be obtained in a lung. It is to be remembered that this charcoal was in an exceedingly fine state of subdivision. Thus, on mixing 0.1 gm. of very finely powdered animal charcoal in 500 c.c. of water, the fluid was only very slightly darkened. One-tenth of a gram of the precipitate from the lung, dissolved in 500 c.c. of water, made the fluid almost jet black in color, although this 0.1 gm. represented only about  $\frac{1}{40}$  gm. of carbon. Again it will be seen that about one-fourth part of the ash was in the form of an oxide of iron. The amount of emery was represented by oxide of aluminum and oxide of silicon. These two together made up about 2.5 + gm., so that considerably over one-half of the ash was in the form of emery, and the emery and iron together made up more than three-fourths of the total amount of the ash.

### *Discussion.*

Dr. PRUDDEN remarked that more than a barrel of water had been made as black as ink by the pigment contained in the lungs of this person. The investigation had an obvious and important bearing on infection through the lung, because it showed how many particles might pass all the safeguards which the air passages present.

### THREE CASES OF DUODENAL ULCER, TWO WITH SYMPTOMS OF ACUTE APPENDICITIS.

Dr. HARLOW BROOKS made this report:

CASE I.—This was a female child, twelve years of age, in good general health up to a few days before death. She had been constipated at that time, and a calomel purge had been given. When it had operated she had



begun to suffer from severe pain, and had died a few hours after. At the autopsy, on opening the abdomen the omentum was natural, but the transverse portion of the colon was distended with gas. In the right hypochondrium was a mass of adhesions, partly recent, involving the hepatic flexure of the colon, gall bladder, duodenum, and pyloric end of the stomach. The common bile duct was occluded by adhesions. The surrounding peritonitis was quite sharply localized. On the internal wall of the second portion of the duodenum was a mass of old adhesions, and opposite this the wall of the duodenum was sacculated and of a purple color. The duodenum contained 2 c.c. of normal chyme. An area of the mucous membrane, one and one-half inches in diameter, was the seat of an erosion. This ulcer was not punched out, and microscopically it gave no evidence of tuberculosis or of anything else indicative of its origin. No other lesion of the digestive tract was found. The parents had been healthy.

CASE II.—This was a Russian, about thirty-eight years of age, who had entered Trinity Hospital with a supposed abscess of the lungs. He had been operated on for this and a large quantity of pus, devoid of odor, had been opened up. Two weeks later an abscess had formed in the groin, and had been incised. Later on there had been symptoms strongly indicative of appendicitis, and the abdomen had been opened. The abdominal cavity contained pus which bacteriologically contained streptococci. At the autopsy, all the operation wounds were in a healthy condition. The abdominal cavity contained about one litre of thick, creamy pus, devoid of odor. There was a sacculated empyema in the pleural cavity, and connected with the pus pocket found in the abdominal cavity. There were many areas of old peritoneal adhesions. The spleen was almost

bifurcated by an infarction. The main pus pocket had been bounded by the spleen, the posterior surface of the pancreas and duodenum, and the posterior abdominal wall. A well-defined abscess cavity occupied almost the entire head of the pancreas, and this had undoubtedly been the original focus. The mucous membrane of the duodenum showed a characteristic punched-out ulcer situated near the pyloric ring; the base had been healed over. A similar lesion was located in the wall of the stomach also near the pylorus, but this was completely perforated. There was no inflammatory action around the ulcers. The liver showed marked interstitial cirrhosis and several gummata of considerable size.

CASE III.—In this case a man, twenty-nine years of age, had been admitted to the J. Hood Wright Memorial Hospital to the service of Dr. LeBoutillier. For the past three years he had had nausea and vomiting in the morning. For two months previous to admission he had been passing a large quantity of dark, irritating urine. He had been told several times that he had had syphilis. Just before coming to hospital he had been on a long drunk. He then developed pain about the umbilicus and vomited clear fluid. On the day previous to entrance he had had a sudden chill. On inquiry it was learned that he had had a somewhat similar, but less severe, attack some years ago. There were moderate distention of the abdomen, and tenderness, with moderate rigidity of the left rectus muscle. The diagnosis of general peritonitis, probably originating from appendicitis, had been made by both his attending physician and the hospital staff, and accordingly the abdomen had been opened. About one litre of fluid containing shreds of fibrin had escaped. The vermiform appendix was swollen and congested. It was amputated, and the stump inverted. The patient



died after about thirty hours. Though the external coat of the appendix was eroded no perforation could be detected, and nothing to account for the peritonitis. At the autopsy, eight hours after death, both œsophagus and trachea showed many tiny healed ulcerative patches. There was general fibroid enlargement of the lymph nodes. The abdominal cavity contained 500 c.c. of slightly turbid fluid, and the entire peritoneum was covered with an even fibrous covering. There was absolutely no odor to the exudate. There had been natural reaction near the field of operation, and the wound and stump of the appendix appeared healthy. Near the cardiac extremity of the stomach were numerous areas of submucous hemorrhage. On the superior anterior surface of the duodenum near the pylorus was a conical perforation with apex extending out to the peritoneum. The ulcer was nearly 1 cm. deep. There were no evidences of acute inflammatory action about the perforation, and the appearance was that of a recent ulcer produced by mechanical means. Sections of the appendix when examined microscopically showed an inflammatory exudate on the outer surface, but in none had it extended through to the endothelium. There was extensive degeneration of the glandular tissue including the glands of Brunner, due to post-mortem digestion or possibly to early post-mortem change.

Ulcers of the duodenum, the speaker said, were very rare. Perry and Shaw, in 17,652 autopsies, had found only fifty cases of duodenal ulcer. It was more common in males. Excluding the cases following burns there were but eight women to forty men. This disproportion between the sexes had never been explained. The ulcers following burns were of a different type from the duodenal peptic ulcers present in these cases. These ulcers probably followed sharply localized nutritional disturbance



in the wall of the gut. They occurred most frequently when there was hyperacidity of the gastric juice. This would seem to be confirmed by the fact that they were rarely found below the first portion of the duodenum. The predisposing causes were not yet known. Perry and Shaw had found twenty-five cases associated with tuberculosis. None of the authorities seemed to consider syphilis one of the causes, yet, in his opinion, this was a very probable cause. It should be noted that two of his subjects were syphilitic. The fact that the symptoms had simulated appendicitis was of clinical interest. It was well to remember that the pus associated with a peritonitis set up by the perforation of a duodenal ulcer was devoid of odor, in marked contrast with the usual condition found in cases of appendicitis. The aspiration of a few drops of pus prior to operation might give assistance in deciding on the true condition present.

The paper of the evening was read by Messrs. FRANK and WEIL; they were introduced by Dr. VAN GIESON. In his introduction he stated that the theory of

### *Neuron Retraction*

had been one of the guiding principles of the researches at the Pathological Institute of the New York State Hospitals for the past three years. In endeavoring to make clear what was meant by neuron retraction it would be necessary to recall briefly the relation between mind and body, between psychomotor manifestations and physiological and pathological processes in the nervous system. The relation between these two sets of phenomena was not, as is too frequently supposed, one of succession, but of co-existence. Dynamic processes in the nervous system were not transmutable into psycho-motor

manifestations. In each domain — in mind and its physical substratum — there were indeed uniformities of succession; but between these two the relation was not that of antecedent and consequent, but of parallelism. The terms neuron retraction and expansion should be applied to pathological processes in the neuron giving rise to catalysis and synthesis of neuron aggregates functionally associated. Concomitant with these physical processes of retraction and expansion of the neurons were psychopathic phenomena which might be termed dissociations and reassociations of consciousness. Correlative with catalysis of neuron aggregates occurring through retraction were the psychopathic waking states; concomitant with the reverse process of neuron expansion inducing synthesis of the neuron aggregates were the psychopathic sleeping states. The processes of retraction and expansion of the neuron were not universal for the whole nervous system. In the lower parts of the nervous system there was no such thing as neuron retraction and expansion; the neuron aggregates were firmly united, are in fact concrescent. It was rather singular to witness the division of investigators in psychiatry and neurology into two hostile camps, the one faction believing that retraction was universal for the whole nervous system, and the other, because some of its objectively minded disciples had demonstrated anatomically annectant neurons in an invertebrate or in some one place in the human nervous system, fell headlong into the belief that the whole nervous system was composed of concrescent neurons, and, what was still worse, that our mental life must be bound to the fixed and unassimilative type of pulses of consciousness. Neither of these views was right. The theory which recognized each of these views seemed to be the only tenable one. The speaker asked to be accorded the privilege of stat-



ing that the Institute where Mr. Weil and Mr. Frank had conducted their investigation was one of the first to recognize this theory, and had fully two years ago declared and insisted on the standpoint that the human nervous system was composed of both free, retractile neurons in the higher cortical spheres, and of concrescent neurons in the lower parts. The speaker desired to correct the apparent misunderstanding that the theory of neuron retraction in the above mentioned Institute postulated universal retractility for the whole nervous system. The scope of these introductory remarks hardly permitted of discussion of the process equivalent to retraction in the fixed and annectant neuron aggregates. There was, however, quite an analogous process. In annectant neurons this process could not be termed retraction, but was disaggregation. The breaches of continuity and their restitution had also parallel psychomotor manifestations which constituted respectively neuropathic waking and sleeping states, and were described in a paper entitled "Neuron Energy and its Psycho-Motor Manifestations." The paper about to be presented was one of three correlated branches of investigation of neuron retraction pursued at the Institute, in which the subject-matter was examined from both the inductive and deductive standpoints. In the paper of Mr. Weil and Mr. Frank, the psycho-motor phenomena concomitant with neuron retraction were entirely discarded and the question was considered from a purely morphological and inductive point of view. In this paper the morphological evidence of neuron retraction as furnished by the Golgi methods was shown to be wholly untrustworthy. In a second paper the argument used was to examine the cytological structure of the neuron, to study its analogy with the structure of other cells endowed with distinct motility, and also to



consider in general terms some of the phenomena of psycho-motor dissociations. In the third paper the argument was largely deductive. Neuron retraction and expansion were assumed. This was taken as a postulate and was verified by the study of abnormal psycho-motor phenomena, especially the psychopathies. This last line of argument furnished the only cogent course of reasoning to prove the retraction theory. The theory could only be substantiated by the study of the phenomena of abnormal mental life by the deductive method, and those who attempted to investigate the question from an entirely inductive and morphological basis could hardly have a broad, intelligent conception of the nature of the problem. We had as yet no actual demonstration of neuron retraction, nor were we likely to have for some time to come. In fact, this was not absolutely essential for the establishment of the theory if neuron retraction was assumed, and if psychopathic phenomena and much in the course of the phylogenetic and ontogenetic evolution of consciousness were explained by this theory, and indeed verified the theory, it was perfectly justifiable to accept it. The great weight of proof of the retraction theory rested not on morphological and physiological bases, but on the study of the living phenomena of mental life. It was this line of study from the psychological and psycho-pathological standpoint that had led us to accept and rely on the retraction theory as a great and powerful guiding principle of our investigations. Those who opposed the retraction theory because it was not proven by tangible and actual objective demonstration might equally well be sceptical of the theory of molecules and atoms and the whole fundamentals of physical sciences. No one had ever seen an atom or a molecule, or had been able to receive any perceptual recognition of them. These things were entirely conceptual and inventions

of the imagination. But these conceptions of atoms and molecules were so universally verified by the phenomena of the physical world that no chemist or physicist hesitated to accept the atomic theory. Neuron retraction and expansion were far too subtle processes for objective demonstration, at the present time at any rate. We ought to bear in mind that the study of living phenomena was just as cogent and important in substantiating theories as laboratory work with lenses, test tubes, and crucibles. The speaker did not want to minimize the objective demonstration of patho-physiological processes in the laboratory. This had been and always would be of incalculable benefit, and rested our theories on a surer footing. But it was quite fitting with one of the greatest and most profound problems in mental and nervous life before us, to remember that the study of the living phenomena might give us a key and a grasp on the question when laboratory study of dead tissues would signally fail. Those who could combine both points of view had indeed the coign of vantage. The history of medicine showed that reflective clinicians again and again had in general terms foreshadowed theories of patho-physiological processes long in advance of their detailed elucidation in the laboratory, and he felt sure that the retraction theory would be established by the explanation of the living phenomena of consciousness in advance of the demonstration of the physical pathway beside which they travelled.

Mr. WEIL then read the paper by Mr. FRANK and himself :

ON THE EVIDENCE OF THE GOLGI METHODS FOR THE  
THEORY OF NEURON RETRACTION.

In the following paper a brief statement is given of the results of studies by the authors during the past three



years in the Pathological Institute of the New York State Hospitals in connection with the theory of neuron retraction. All the historical evidence which has hitherto been advanced in support of the theory has been based upon the Golgi method; the object of the present research is an experimental investigation of the validity of certain histological changes in the processes of the nerve-cell, supposed to be indicative of neuron retraction.

The literature of the subject is extensive and will not be detailed in this place; a complete *résumé* is furnished by W. Ford Robertson in the last number of the English magazine *Brain*. In brief, neuron retractility is by the majority of observers regarded as a function of the protoplasmic processes, and is best manifested by the pyramid cells of the cerebrum and the Purkinje cells of the cerebellum. Two changes are said to occur: First, the appearance of localized swellings, or "varicosities," along the course of the dendrites, and, second, the disappearance of the gemmules, or dendritic spines, at the site of these swellings. The two phenomena are supposed, except by Lugaro, to be correlated: the spinous processes withdraw into the body of the dendrite, and, by so doing, produce a localized swelling. Human and experimental material of the greatest diversity has been investigated—of the former, brains of diphtheria, typhoid, insolation, etc., of the latter, brains of animals poisoned by arsenic, lead, morphine, strychnine, chloroform, tuberculosis, hydrophobia, experimental strumapriya, "experimental uræmia," and so forth. The method employed has always been the rapid Golgi (or Cajal) method, except in the case of certain recent work of Lugaro's, which has employed Cox's modification of the corrosive method. Criticism of the conclusions above stated has not been lacking. Both gemmules and varicosities are by some authorities considered to be



artifacts. By others, *e. g.* Lugaro, while their occasional authenticity is admitted, a valuable proportion of their number is attributed to post-mortem processes; Lugaro asserts that the only form of the Golgi method which does not exaggerate the true number of varicosities is the Cox modification.

In this investigation, four forms of the Golgi method were made use of, namely, the rapid, mixed, and slow modifications of the bichromate-silver method, and the Cox modification of the corrosive method. The number of animals used was forty-three. There were five cases of human material, three adults, and two foetuses; one dog, and thirty-seven rabbits. Of the rabbits ten were normal; of the remainder, two were poisoned by morphine, one by strychnine, four by chloroform, and the rest by the injection of hypertoxic urine or serum. Nine of the rabbits were treated uniformly according to four methods, the three bichromate-silver modifications and the Cox; two were treated according to the rapid Golgi method and the Cox; the rest according to the rapid, mixed, or slow procedure alone. In all, 342 pieces were sectioned. The cerebral cortex alone was studied.

The conclusions reached are the following :

1.—The same material, when treated by different methods, yields different results. The nature of the differences in case of each kind of material is as follows :

All material treated according to the slow method of Golgi, shows, as a rule, an almost absolute freedom from varicosities; varicose cells occasionally occur, but with a relative frequency which is perhaps not greater than a fraction of one per cent. of the total number of pyramid cells impregnated. Exceptionally, a large proportion of varicosities occurs.

The mixed method and the rapid method may be considered together; these two methods yield practically

similar results as regards the varicosities and the gemmules. The gemmules are almost invariably present and generally regular, provided the dendrites have taken the impregnation. The varicosities occur in variable proportions, although their frequency regularly is greater, and almost always very much greater, than is the case in the slow method. In some sections, almost every dendrite is varicose, in others, hardly any.

In the Cox method, a fair amount of varicoseness is generally present at any stage of fixation. Gemmules are almost universally present and regular.

2.—The above results are independent of the nature of the material, whether normal or toxic. Normal material, as well as toxic, is, as a rule, free from varicosities when treated by the slow method. Normal material, as well as toxic, exhibits a variable amount of varicosity, when treated by any of the other three methods which we have used. We find that it varies within exactly the same limits as the abnormal, that every degree of varicoseness can be illustrated with equal freedom from either, and, finally, that it is impossible for an unprejudiced observer to differentiate or distinguish between the two kinds of material.

3.—The same material does not yield constantly identical results, when treated by one and the same method. Pieces from the same animal, when immersed in the same fluids of the slow, mixed, rapid, or Cox method, may illustrate the extremes of varicoseness produced by that method.

The above conclusions seem to demonstrate that the varicosities are to be regarded as artifacts of the Golgi method.

#### *Discussion.*

Dr. JOSEPH COLLINS said that he had been specially



gratified with the paper and its very convincing arguments. The fact that varicosities had appeared on other cells than the pyramidal cells had always been a great stumbling-block to those who had put forward facts from morphological experiments. He hoped to be able, at some time, to controvert most of the statements made by Dr. Van Gieson. He would not think of accepting his definition of the retraction theory.

Dr. E. D. FISHER said that it was interesting to learn from these experiments that these varicosities were the results of the methods employed, and had no special significance. He had always noted that Dr. Van Gieson took special pains to show the true nature of many of these phenomena, whereas other observers had seemed disposed to theorize on insufficient ground. Perhaps the most convincing argument presented was the statement that the same appearances were presented by material from both normal and abnormal animals. He agreed with Dr. Van Gieson that a theory, no matter if it proved ultimately false, was of great advantage in studying a subject like psychology.

Dr. PRUDDEN said that he had been deeply interested in the paper because it demonstrated the great value of careful technical procedure. He did not conclude, however, as another speaker had seemed to do, that this work should be regarded as evidence against the retraction theory, but rather that this sort of evidence was not good evidence, and that the retraction theory still remained, in so far as this study goes, neither proved nor disproved.

Mr. WEIL said that by a number of methods it was impossible to demonstrate the presence of gemmules, but it was perfectly possible to demonstrate them by other methods. He was of the opinion that the majority of workers with the Golgi method were coming to regard



the gemmules as a legitimate product. They appeared regularly and only on the cortical cells.

Dr. VAN GIESON said, in response to Dr. Collins, that when different degrees of conduction in pathological conditions were plainly evident in one and the same set of neurons, it was difficult to give any simpler explanation, and one more in accordance with our knowledge of the physiology and morphology of the neuron, than retraction.

---

*Stated Meeting, December 13, 1899.*

W. H. PARK, M.D., VICE-PRESIDENT, IN THE CHAIR.

A CASE OF ELEPHANTIASIS OF BOTH EARS.

Dr. J. H. LARKIN presented for Dr. HODENPYL the specimens and photographs from a case of elephantiasis of the lobes of both ears, apparently produced by the irritation arising from the wearing of earrings. The removed portion measured 1.4 by 4 by 2 cm., and it had been taken from a lady aged thirty-seven years who was in good health. She stated that they had been enlarging for the past year and a half. Microscopical examination showed hyperplasia and a moderate exudate in the neighborhood of the blood-vessels. The speaker said that while elephantiasis of the lobes of the ears from wearing earrings was not unusual, the increase in size in this instance had been excessive.

PAPILLOMA OF THE LARYNX ; SUFFOCATION.

Dr. JAMES EWING presented the larynx taken from a woman, forty years of age, who had apparently been in good health until about ten days before death, when she had begun to suffer from very severe dyspnoea. She had been seen at the dispensary of Bellevue Hospital, and it had been noted that the obstruction to

breathing was extreme. The woman had been admitted to the medical side of the hospital, but no definite diagnosis had been made. The peculiar character of the dyspnoea had led one of the house staff to suspect obstruction in the trachea. The laryngoscopic examination was negative, but had to be performed under difficulties. She had died in a few hours. The autopsy had shown the organs normal with the exception of an extensive chronic diffuse nephritis. On opening the larynx, just below the vocal cords was to be seen a gray and apparently necrotic mass, about 2 by 3 cm., attached by a small pedicle to the mucous membrane below the right ventricle posteriorly, and lying somewhat loose in the cavity of the larynx. It had caused an acute catarrhal inflammation in the upper half of the larynx and a necrotic inflammation of the mucosa immediately surrounding it. It almost completely obstructed the air passages, and fully explained the cause of death. It had been difficult at first to determine the nature of this body. It had been necrotic throughout, but on staining it in various ways remnants of an epithelial layer could be made out, and, in general, the outlines of a papilloma. The diagnosis, therefore, was papilloma of the subglottic mucosa; necrosis of this papilloma and suffocation. The speaker said that one or two laryngologists had seen this case before a microscopical examination had been made, and they had stated that papilloma of the larynx never caused death in this way. If this was true, the case must be looked upon as unique.

#### *Discussion.*

Dr. JONATHAN WRIGHT said that if this growth of the larynx was simply a papilloma, the case was, so far as he knew, entirely unique. In tubercular and

syphilitic lesions there were apt to be papillomatous growths all over the mucous membrane of the larynx. Sometimes on removing these there would be left a deep ulcer beneath, and very frequently with syphilis there would be an involvement of the cartilage. He had seen these papillomatous masses in a case of tuberculosis of the larynx so marked that laryngectomy had been done and the inside of the larynx thoroughly scraped out, with the idea that the patient would recover with a sound larynx. Unfortunately the patient had died as a result of an accident. He would be very sceptical indeed as to a non-specific condition in the case just reported; it would be interesting to have sections made immediately around the ulceration and deeper down in the tissues with the object of searching for syphilitic lesions. The history pointed very strongly to syphilis of the larynx. Such patients often presented for a long time few, if any, symptoms until the onset of dyspnoea, which would then increase so rapidly that unless the treatment was prompt and effective the patient would be liable to die. A recent case was cited in which, although the dyspnoea had not been urgent, he had sent the patient to the hospital. The patient had, however, lost the note, and consequently there had been some delay. As a result, the man had nearly died before tracheotomy could be done. In that case there had been nothing but an abscess of the larynx of syphilitic origin. After having had an opportunity of examining macroscopically the specimen of papilloma of the larynx, Dr. Wright was ready to admit that he could discover no evidence of a syphilitic or tuberculous base to it. He had seen several cases of tuberculosis at the base of the epiglottis; while not very common they were not extremely rare. They were occasionally mistaken for syphilis, but the finding of the tubercle bacilli would easily settle that question.



## TUBERCULOUS ULCER AT THE BASE OF THE EPIGLOTTIS.

Dr. EWING also presented a larynx from a case of chronic miliary tuberculosis. It exhibited an ulceration of considerable extent at the base of the epiglottis. There were no gross lesions of the mucosa, but the entire base of the epiglottis was infiltrated with tuberculous tissue, and the base itself was the seat of an ulcer about 3 by 1 cm. Tubercle bacilli were found in this ulcer. This was a rather rare localization of tuberculosis of the upper air passages.

## GENERAL MILIARY TUBERCULOSIS ; THROMBOSIS OF SUPERIOR MESENTERIC ARTERY.

Dr. EWING next exhibited specimens taken from a girl aged seventeen years whose health had been failing for some months before her death. The chief symptom had been an irregular diarrhœa. About ten days before death this diarrhœa had been more severe, and had been associated with abdominal pain. The temperature had then risen, and the case had afterward followed the usual course of general miliary tuberculosis. At the autopsy the colon had been found to be the seat of a few rather old and not very extensive tuberculous ulcers, which were partly healed. There were also an acute catarrhal inflammation of the ileum and jejunum, and a considerable swelling of the lymphatic tissues throughout the lower ileum, but there were no tuberculous ulcers of the small intestine and no involvement of Peyer's patches. Beneath the stomach was a large mass partly adherent to the adjoining viscera. When dissected out it was found to consist of the mesentery in a state of extensive bloody infiltration. There were thrombosis of the superior mesenteric artery, and

suppurative arteritis with miliary tubercle surrounding the vessel, but not involving the clot. Besides the lesions of general miliary tuberculosis in this case the lymph nodes at the root of the lung were very slightly involved; on the side of the neck were one or two which were slightly caseous. With the exception of the chain in the neck the chief seat of the lesions was the upper mesenteric region. The infection had probably arisen from the initial ulcers in the large intestine, and had lodged in these particular lymph nodes, and death had resulted from infection of the superior mesenteric artery, dissemination of the bacilli through this artery, and a complete general miliary tuberculosis.

*Discussion.*

Dr. W. H. PARK asked if Dr. Ewing felt fairly confident that he could usually detect the primary lesion in cases of tuberculosis. He thought there might be cases of incipient pulmonary tuberculosis in which bacilli were swallowed, and thus intestinal lesions started, making it difficult to determine which lesions occurred first.

Dr. J. S. THACHER asked if there had been no further appearance of interference with the circulation of the intestine as a result of the cutting off of the superior mesenteric artery; under such circumstances there were usually very striking necrotic changes.

Dr. EWING replied that whatever syphilis might have had to do with the first case of papilloma, he could not understand how a constitutional disease could produce a mass of this sort in the trachea. If syphilis was present it must have been entirely secondary as a cause of death. The anatomical condition was such as could hardly have been produced by syphilis. An interesting feature of the case was the absence of cyanosis and the presence of



extreme pallor in spite of the marked obstruction in the trachea. Several cases which had been presented to this society seemed to emphasize this fact, namely, that slow asphyxia was apt to be associated with extreme pallor and not with cyanosis. It was, of course, impossible to say definitely which lesions had developed first, but in the case presented he thought it was highly probable that the intestinal tuberculosis had been primary. With regard to the condition of the intestine as a result of the thrombosis of this large vessel, he said that it was a matter of surprise that the changes had been so slight. There had been no areas of necrosis and no ulcerations, although the blood content of the intestinal wall was considerably increased. The thrombosis was complete, and partly organized.

#### A CASE OF CARCINOMA OF THE ANTRUM OF HIGHMORE.

Dr. LEON T. LEWALD presented specimens from a man forty-five years of age. The condition had not been diagnosed before death; indeed, it was difficult to detect the presence of these tumors in the antrum of Highmore. The autopsy had disclosed a tumor obstructing the nasal fossæ, and growing apparently from the left antrum of Highmore into the nasal fossa on that side. From thence it had apparently extended upward through the sphenoid and ethmoid bones into the skull in the anterior fossa, and had produced a growth measuring about 2 by 1.5 cm. This had pressed on the left frontal lobe. The growth had also passed through the orbital plate of the frontal bone and back through the sphenoidal fissure into the middle fossa of the skull, producing a growth measuring 1 by 0.5 cm. This had pressed on the temporo-sphenoidal lobe of the brain. The tumor had also extended downward through the hard palate and had involved the mucous membrane of the mouth, and



extending down the pharynx had involved the lymph nodes of the neck and even the bronchial lymph nodes. There were also growths in the lungs, apparently of a metastatic nature. Two nodules were also found in the upper surface of the liver. The primary growth appeared to have started from the left antrum, although there was some difference of opinion on this point. A number of sections of the tumor had been examined by Dr. Dunham, and had been found to be carcinoma.

### *Discussion.*

Dr. E. K. DUNHAM said that the chief points of interest in this specimen were in connection with the lesions of the lung, where the neoplasm seemed to have appropriated the stroma of the lung tissue for its own purposes, and not to have developed any stroma in that organ. The microscopical examination had not been completed, but the specimen appeared to be one of medullary carcinoma of poor nutrition, there being large areas of necrosis. In the brain it had had certain peculiar anatomical relations with the blood-vessels. Sections of the growth were exhibited under the microscope.

Dr. IRA VAN GIESON asked if the hypophysis or the optic tracts had been destroyed.

Dr. LEWALD replied that it had not involved either of these regions.

Dr. JONATHAN WRIGHT said that there were two or three cases on record in laryngological literature which had begun in the antrum of Highmore. One of these he had seen clinically and had examined microscopically. There had been some doubt regarding the nature of the growth. It was a polyp of the antrum which had gone down through the tooth socket and had appeared

in the mouth. The patient had been between forty-five and fifty years of age. Thinking it to be simply a polyp an operation had been undertaken, but it had been found that the antrum was filled with grumous material. The tumor had been about the size of a black walnut, perfectly round, and having a rather large pedicle. It had evidently filled the whole antrum. Thick sections had been made through the whole polyp from one side to the other, and deep down on the stroma it had presented a perfect picture of epithelioma. Dr. Prudden had confirmed this diagnosis of epithelioma. The case had been followed for over a year, and there had been no recurrence. This, and one other case in literature, were the only ones he knew of in which it had been definitely stated that the growth had originated in the antrum of Highmore. It was quite possible that more cases were on record, as most of them came under the observation and care of the general surgeons.

#### A CASE OF CONGENITAL ATRESIA OF THE DUODENUM.

Dr. J. S. THACHER showed a specimen in which the small intestine appeared to be the continuation of the common bile duct. It had been taken from a child who died at the age of six days, having been operated upon two days before for an imperforate anus. The child had vomited all food. No communication could be found between the duodenum and the stomach.

#### EXTREME STRICTURE OF THE URETHRA WITH SECONDARY CYSTITIS AND HYDRONEPHROSIS.

Dr. THACHER also exhibited specimens from a case in which the immediate cause of death had been suicide from carbolic acid poisoning. The autopsy had, however, revealed extensive disease of the genito-urinary

•

tract, and particularly a very narrow stricture of the urethra. An opening about the size of the shaft of a pin had been found after some searching, in the centre of an oblique membrane which had occluded the urethra. There had been no clinical history, but it had been assumed that the membrane was of a cicatricial nature.

Dr. MOSCHOWITZ remarked that it was certainly not such a stricture as would arise from traumatism or from gonorrhœa, and consequently he would like to know more regarding its nature. Might not this stricture be congenital?

#### *Discussion.*

Dr. THACHER said that he had, perhaps hastily, assumed the stricture to be the result of inflammation, or possibly of inflammation aided by trauma.

#### DOUBLE SPLEEN.

Dr. LEWIS A. CONNER reported a case of double spleen and exhibited the specimens. Each spleen measured approximately 8 by 5 by 2.5 cm., and weighed 60 grams, and each had its own branch of the splenic artery, and was, in other respects, apparently normal. Of course, a small supernumerary spleen was very frequently met with, but he had never before seen two spleens of the same size.

#### A CASE OF PERFORATING DUODENAL ULCER.

Dr. CONNER also presented a duodenal ulcer removed at an autopsy made a few hours previously. The patient had entered the Hudson Street Hospital with symptoms closely resembling those of appendicitis.

•



Dr. Bolton had made the usual incision for appendicitis, and had found the right side of the abdomen filled with a thin, purulent secretion, and the appendix normal. Through a second incision the pus had been seen to come from the region of the liver and diaphragm. There had been no evidence of gas in the peritoneum at the time of operation. The man had lived for nearly a week after operation. The autopsy showed a severe peritonitis localized around the cæcum and along the ascending colon, but there was no general peritoneal infection. An old oblong ulcer was found immediately below the pylorus, which had had the peritoneal coat for its base for some time, and which had finally perforated by a minute opening. Near this was what appeared to be the scar of another ulcer. There was a history of a similar attack four years ago. Surgeons were beginning to realize that, in general, the higher up the perforation the milder the infection and the better the prognosis, and the case just reported seemed to bear out this view. In this case there had also been a croupous inflammation of the cæcum and ascending colon—in other words, that part of the intestine adjacent to the infected area of peritoneum.

#### *Discussion.*

Dr. PARK said that this idea about the mildness of the inflammation in cases of high perforation was new to him and most interesting. In the duodenum there were usually but few bacteria, and those largely from the food which had escaped the deleterious action of the gastric juice while in the stomach.

Dr. HARLOW BROOKS said that at the last meeting he had presented three cases of duodenal ulcer, and was therefore particularly pleased to observe the close

similarity between this case and his own. The presence of a non-odorous pus, and of symptoms of appendicitis, made the similarity very close, and emphasized again the importance of the character of the abdominal exudate as indicating the presence of duodenal ulcer.

Dr. CONNER said that he had been informed that interesting bacteriological investigations had recently been made by Dr. Cushing at the Johns Hopkins Hospital regarding the intestinal tract, and this had suggested the line of thought just presented.

Dr. THEODORE JANEWAY asked if perforating ulcer of the stomach did not often produce gas abscess underneath the diaphragm, with the production of very foul pus. He had seen such a case last year, in which the first symptom of ulcer of the stomach had been perforation. The woman had gone into collapse and had developed a gas abscess. This had been operated upon in four days, and she had lived a week subsequently. The autopsy had shown a very general peritonitis, but more especially localized in the region of the diaphragm, liver, and stomach. The pus had had a very foul odor, and the gas had formed rapidly even after aspiration. In another case which he recalled there had been associated with severe pain in the upper part of the abdomen, and collapse, the development of friction sounds over the spleen and evidence of a slow abscess formation. After about one hundred days the abscess had burst into the left pleural cavity, and the pus had been found to contain only streptococci. About forty days later, when apparently doing well, she had died with symptoms of secondary abscess of the brain.

Dr. E. LIBMAN mentioned a case of perforation of a gastric ulcer associated with thrombosis of the upper branch of the splenic vein. There were infarction and gangrene of the upper half of the spleen, with very foul



pus and gas in the subphrenic space. Cultures showed streptococci and the bacillus proteus vulgaris. With reference to the greater virulence of perforations in the lower part of the intestine, he referred to an investigation made by him some years ago on streptococcus infections of the bowel, as a result of which he had found that the streptococci increased in number from the duodenum down, and had been most numerous in the cæcum and lower part of the ileum, and that the changes had been most marked in the latter regions. This was possibly due to greater stagnation in these places.

CARCINOMA OF THE LIVER SECONDARY TO A SMALL MAMMARY CANCER.

Dr. GEORGE P. BIGGS presented a specimen taken from a woman, sixty-five years of age. No history of the case could be obtained. The liver was found at autopsy to be greatly enlarged and to contain an enormous amount of carcinomatous tissue (chiefly in the form of nodular masses 1 to 3 cm. in diameter). A careful search through the abdomen and thorax failed to show any other carcinoma. Finally an examination was made of the breasts, both of which were quite small and of equal size. There was nothing in their outward appearance to suggest the presence of a tumor, but on palpation the right breast was found to be very firm, and incision revealed a scirrhous carcinoma involving the whole mammary gland which measured but 6 by 5 by 15 cm. Metastatic deposits were found in the right axillary glands. Microscopical examination showed a marked similarity in the type of cells found in the tumor tissue from the breast and the liver, and suggested the probability that the involvement of the liver was secondary to that of the breast. Additional evidence in support



of this conclusion was to be found in the fact that it was the right breast which was involved, and that from this tumor metastasis had occurred in the axillary glands. It was of course possible that there might have been independent development of tumor tissue in each organ but this seemed improbable. The case served to emphasize the importance of great thoroughness in making post-mortem examinations.

*Discussion.*

Dr. VAN GIESON raised the query as to why the tumor in the liver should have grown to such an enormous extent while the carcinoma of the mammary gland had apparently ceased growing.

Dr. BIGGS replied that he had no explanation to offer. There did not appear in the sections to be any association of the growth with the bile ducts.

Dr. EWING commented upon the gross appearance of this liver. He asked if there had been any distinct indications of secondary growth, or if it had been uniformly distributed as in the section exhibited.

Dr. BIGGS replied that it had been uniformly distributed.

A CASE OF FOREIGN BODY IN THE APPENDIX SIMULATING  
GALL STONE.

Dr. J. H. LARKIN presented a specimen removed from a man, fifty-four years of age, who had given a clinical history of repeated attacks of gall-stone colic for a number of years. About three days before admission to hospital there had been an unusually severe attack of pain, accompanied by fever and depression. He had only been in the hospital an hour or two when the symp-

toms had become very urgent, and the man had died before he could be operated upon. At the autopsy forty-one gall stones had been found in the gall bladder, and in the intestine three stones. The common duct had been greatly dilated, and there had been evidence that a number of large stones had passed through the duct at different times. The vermiform appendix was found twisted upon itself, and turned upward and backward. The middle portion was free, but the tip was adherent to the cæcum and could not be separated. On opening the appendix it was found that the tip of the appendix had perforated into the cæcum, and projecting through this opening was a stellate-shaped stone. On slitting up the appendix farther he had found three stones—one in the shape of a clover leaf and the other two with facets, one having six and the other four. Chemical examination of these stones from the appendix showed them to be simply enteroliths and not gall stones. They differed from ordinary enteroliths in their shape and in the presence of facets. The lumen of the appendix had been entirely obliterated, and microscopical examination had revealed a complete replacement fibrosis of the lumen, there being no remains of epithelial or glandular tissue. Mitchell had made a rather recent contribution to this subject, and had collected fourteen hundred cases of foreign bodies in the appendix. Out of this large number, cases of gall stones in the appendix had been reported by only three observers.

#### *Discussion.*

Dr. CONNER thought it not impossible that these stones from the appendix were really gall stones, and the fact that one of them presented six facets seemed to point very strongly to this stone having been associated with

a number of other stones. He suggested that such stones might lose some of their chemical characteristics after having been for some time in the bowel.

Dr. LARKIN replied that in the chemical reactions of the enteroliths in the appendix cholesterin had not been found. The appendix itself showed that a chronic process had been going on for a long time. The chemical examination had shown very definitely that these stones in the appendix were not gall stones. The three stones present in the appendix might have produced a large number of facets. The formation of these facets seemed to him to be the result of muscular action.

Dr. THEODORE JANEWAY asked if the chemical examination had been made from the exterior, or whether it had been made on the interior, or by section.

Dr. LARKIN said that quite a large piece of one of the stones had been chipped off and subjected to chemical examination.

#### COLONIES OF THE PLAGUE BACILLUS.

Dr. W. H. PARK showed an agar culture and stained preparations from agar and bouillon cultures of the plague bacillus obtained from the cases of bubonic plague found on the steamer *J. W. Taylor*. He said that although full half a cubic centimetre of pus had been extracted from the buboes of the ship's captain and cook, only three to five colonies had developed in each case. The rapid disappearance of the bacilli at the time of the pus formation in the buboes had been noted by the earlier observers. Their form, cultural and staining characteristics were identical with those of two cultures obtained from that disease which had been brought to him from India.

Dr. ISAAC LEVIN read a paper on



## MUCINÆMIA, AND ITS RÔLE IN EXPERIMENTAL THYRO-PRIVA.

It is a well-established fact that an organism deprived of its thyroid, either through disease (myxœdema, cretinism), or through an operative removal of the gland, is suffering from some kind of an auto-intoxication. Some substance, which has either been previously transformed by the cells of the thyroid, or else neutralized by some other substance produced by the gland, accumulates in its absence in the blood and poisons the organism. But what is the nature of the substance or substances? This question has hardly been approached yet experimentally in a direct way. The discovery of an increased amount of mucin in the tissues in myxœdema as well as in thyroidectomied animals led Horsley and others to suppose, *a priori*, that the symptoms of cachexia thyropriva may be due to accumulation in the blood of mucin, which is normally transformed by the thyroid. It seemed to me all the more desirable to test the matter experimentally, as I was unable to find in the literature any physiological or pharmacological study of the influence of mucin on an organism. I shall limit myself here to an epitome of my work. A more detailed account of it, together with the tracings and other experiments bearing on the subject, is now in course of preparation and will appear elsewhere.

In order to study the relation between mucinæmia and the thyroid I availed myself of the fact that rabbits stand thyroidectomy a great deal better than carnivorous animals. Until lately it was universally accepted that thyroidectomy was not fatal to rabbits. Gley in his work endeavors to show that the operation is fatal to rabbits if all the parathyroids are also entirpated. But even with him only a small percentage get an acute cachexia, and these die not later than within three days

after operation. The rest either recover entirely or emaciate and die a few months after the operation. I have obtained identical results in my simple thyroidectomies on rabbits. A few of the animals die within twenty-four to forty-eight hours, but by far the greater part survive. Taking this as a basis I did the following experiments: A solution of mucin in one per cent. sodium carbonate was injected hypodermically into eight normal and nine thyroidectomied rabbits. The former remained healthy, while of the latter only one survived. Some of the thyroidectomied rabbits died within forty-eight hours after the injection, while they had previously survived the thyroidectomy from eleven to twenty-five days. Further to study the influence of mucin on the nervous system of a normal animal, I examined the influence of an intravenous injection of a mucin solution on the blood pressure of the dog. The experiments showed uniformly a fall of the blood pressure, even after both vagi and the splanchnic nerve were cut. Subsequent stimulation of the splanchnic again increased the blood pressure. The fall was consequently due to the direct depressing action of mucin on the vasomotor centre in the medulla.

The conclusion to be drawn from this work is, that mucin accumulated in or introduced into the blood of a normal organism produces a certain depressive effect upon the central nervous system; that it is not fatal to a normal organism, and is decidedly fatal to one deprived of its thyroid. Mucinæmia then, one may conclude, is the pathological condition of an organism resulting from the absence of the thyroid function; but this conclusion does not exclude the possibility of other abnormalities arising from the same cause.

This was followed by a second paper by Dr. P. A. LEVENE on



THE CHEMICAL RELATIONSHIP BETWEEN MUCOID, COL-  
LOID, AND AMYLOID SUBSTANCES IN NORMAL  
AND PATHOLOGICAL TISSUES.

The morphological study of diseased organs reveals frequently the presence in the cells and tissues of the latter of substances which do not occur in the same places of the normal tissues. The nature of the substances can be detected by microscopical investigation only very rarely. The microscopical technique is inadequate to disclose the chemical nature of most of the "pathological substances," and they are then identified by their physical properties. And yet only a thorough knowledge of their chemical constitution can elucidate the process of their formation and their relationship to the normal cell constituents.

The substance predominating over any other one in the protoplasm, and most peculiar to it, is one of a proteid nature. It is, therefore, natural to expect the most changes in the proteids of the tissues, when the latter are affected by some disease. Such is, in fact, the case. Pathologists have described several forms of pathological transformations of proteids in tissues under the name of coagulation necrosis, amyloid, colloid, mucoid, hyaline substances, etc. Originally but one distinction between the latter substances and the physiological proteids was detected; while the normal proteids were found to be in the tissues in a state of solution, the "pathological" ones were coagulated. In all other respects they were similar to any proteid material. Amyloid, colloid, mucoid, and hyaline substances were for certain physical properties classified under a special group of "colloidal" substances. The studies on mucin and allied substances, however, soon disclosed that the latter were not simple proteids; that the proteid was



combined in them with a reducing substance, a "carbohydrate" or "animal gum." Thus mucoid and colloid were necessarily regarded as substances distinct from the other colloidal substances, amyloid and hyaline.

Later it was discovered that amyloid was also not a simple proteid. As mucin and mucoid, so amyloid contained a substance capable after certain treatment of reducing Fehling's solution. There was, however, a pronounced difference between mucoids and amyloid: while in the former the "animal gum" was supposed to be combined directly with the proteids, it was in the latter combined directly by the aid of sulphuric acid—in a word, in amyloid the substance capable of reducing Fehling's solution was described as chondroitin-sulphuric acid.

On the ground of the latter discovery three different forms of colloidal substances had to be established viz.: (1) Mucoid and colloid; (2) amyloid; (3) hyaline. However, certain chemical and tinctorial properties of mucin justified to my mind the supposition that the latter must have contained in its molecule an acid radical. To find the acid radical of mucin was the object of this investigation. The work was begun on tendo-mucin, and it was soon found that, similar to amyloid, the mucin was a compound of proteid and of an ethereal sulphuric acid. Further, it was found that, similar to chondroitin-sulphuric acid, the ethereal sulphuric acid of the mucin was nitrogenous, and that it yielded, similarly to the former, chondrosin. With the same object in view submaxillary mucin and colloid of a colloidal carcinoma were analyzed. These two substances were also found to be compounds of a nitrogenous ethereal sulphuric acid.

Thus it seems that the acid radicals of amyloid, colloid, and mucoid substances are very similar to each

other. The investigation into the question whether the substances are only similar or identical is now in progress.

*Discussion.*

Dr. JAMES EWING asked what had been the result of the examination of the blood in cases of thyroprivia as regards the presence of mucin.

Dr. I. LEVIN replied that it was exceedingly difficult to detect such small quantities of mucin.

Dr. VAN GIESON said that these papers, and particularly the second one, were interesting in showing what it was possible for the physiological chemist to do in the way of aiding the pathologist. One more link would be added to the proof of the presence of mucin in the system. If the animals deprived of thyroid, after having been injected with mucin, had been supplied with artificial thyroid extract it should have, theoretically at least, prolonged their lives.

Dr. I. LEVIN replied that the trouble in experimenting in this way with the thyroid extract was, that this extract was of a most uncertain nature. The advantage of working with mucin was that one knew the nature of the substance with which one was working.

---

*Stated Meeting, January 10, 1900.*

T. MITCHELL PRUDDEN, M.D., PRESIDENT.

A CASE OF CONGENITAL RENAL MALPOSITION WITH ANOMALOUS ARTERIAL SUPPLY.

Dr. HARLOW BROOKS presented a specimen taken from a negress, twenty-eight years of age, who had been healthy previously, and had borne one child at full term



without any uræmic or other complications. About one year later she again became pregnant. When supposed to be about three months pregnant she entered the Harlem Hospital complaining of cramp-like pains in the region of the appendix. She had no fever, and palpation at McBurney's point did not give the characteristic tenderness or sensation to be expected in appendicitis. The urine had not been examined until rather late in the case, and then had been found to contain a small quantity of albumin, and some blood corpuscles. There had at this time still been no elevation of temperature. Pelvic examination by the house surgeon had shown the woman to be pregnant, and had revealed the presence on the right side of a mass supposed at the time to be a cystic ovary or a pyosalpinx.

The woman had been rather irritable, but had gradually passed into a comatose condition, when a diagnosis of uræmia had been made. The autopsy revealed a twin pregnancy at about the third month. One umbilical cord was tied in a hard knot. The uterus was in the normal position. The left kidney was about in normal size, but exhibited an acute hemorrhagic nephritis. The right kidney was found below the brim of the true pelvis, and the enlarging uterus had forced it up against the bones of the pelvis. The tissue of this kidney was extremely anæmic. There were two renal arteries on this side, one given off just above the bifurcation of the abdominal aorta into the common iliacs; the other branch was derived from the sacra media. The enlarging uterus had compressed and rotated the kidney, cutting off the blood supply from the sacra media, and also from the upper renal artery, which was given off slightly above the sacra media. It was this that had caused the anæmia of the kidney and probable loss of function. This sudden



increase of function explained the pain and the rather sudden onset of the acute nephritis on the other side. This position of the kidney in the pelvis was not particularly rare, and it was not extremely rare for the sacra media to become the renal artery, but in all such non-functioning cases found in the literature the kidney had been apparently deprived of its function. In none of these other cases moreover, had any symptoms been produced. In the case just reported the malposition of the kidney had probably caused death. In her first pregnancy the uterus had risen out of the true pelvis before it had been large enough to compress the kidney; in the last pregnancy, however, twins being present, the uterus had enlarged sufficiently to compress the kidney before it rose from the true pelvic cavity. The only other interesting feature of the case was the general smallness of the arteries of the body.

*Discussion.*

Dr. HODENPYL asked if the entire blood supply had been cut off from the kidneys.

Dr. BROOKS replied that he thought it had been for the reason that the pelvic blood-vessels had been pretty well filled, and the renal vessels apparently bloodless. The reason that gangrene had not taken place was that the nipping of the kidney had apparently taken place only about three days before death, *i.e.*, at the time she had complained of the cramps.

SOME UNUSUAL CASES OF LEUKÆMIA, WITH DEMONSTRATION OF STAINED SPECIMENS OF THE BLOOD.

Dr. THEODORE JANEWAY reported five cases of lymphatic leukæmia, four of which had been seen in his father's

consulting practice. The fifth had been seen recently in dispensary practice, and he had been able to obtain a partial autopsy. Two of the cases should be classified as acute leukæmia. The first case occurred in a man aged thirty-eight years, who had had severe malaria two years before the onset. There had been a prodromal period of two weeks, during which he had felt tired. The onset had been marked by severe pain in the left side, and during the first week there had been uncontrollable epistaxis. Later on petechial spots had appeared on various parts of the body. There had been some rise of temperature. When seen, four weeks after the commencement, there had been marked pallor; an enlargement of the spleen down to the umbilicus; slight enlargement of the cervical and inguinal lymph nodes, and slight enlargement of the liver. The blood count showed 840,000 red cells and 77,000 white cells. In the stained specimens it was evident that fully ninety per cent. of the white cells were mononuclear cells without granulations. There were a few myelocytes, megalo-blasts, and normoblasts. No mast cells had been found. Death had occurred about two weeks afterward, the total duration of illness having been six weeks and a half. The second case was that of a child aged four and a half years, who had been seen in the University clinic. There had been diphtheria and scarlet fever two years before, and these had been followed by suppurative otitis. As the child had been on Long Island it had been treated at first for malaria. Six weeks before death there had been enlargement of the abdomen with considerable tenderness of the left side. Two weeks later there had been a hemorrhage from the nose and bowel. The blood count had not been made. When seen by the speaker there had been marked pallor of the skin and mucous membrane, with a lemon tint to



the skin. The temperature was  $101^{\circ}$  F., and the pulse was very rapid. The spleen was enlarged down to the umbilicus, and extended well around into the flank. The liver was one finger's breadth beneath the free border of the ribs. The cervical and inguinal lymph nodes on the left side had been very slightly enlarged. The ophthalmoscope had shown a pale retina with numerous large, red-blotched hemorrhages. The blood examination showed very many large red cells present, but scarcely any poikilocytosis. The proportion of white cells was about one in twenty. There was a total of ninety-seven per cent. of lymphocytes. There was a little over two per cent. of polynuclear cells. There were also a few myelocytes, megaloblasts, and normoblasts. One week before death there had been persistent vomiting and bleeding from the gums and from the nose. At this time the abdomen diminished markedly in size. The child had died four months from the beginning of the attack, and six weeks after the onset of acute symptoms. The abdominal organs had been removed the day after death. The spleen only reached to the free border of the ribs, and had shrunk more than one half in the last week of life. Both the liver and kidneys were extremely pale, and the surface of the latter was covered with numerous hemorrhages. The marrow of the rib was pink. He had been unable to obtain the shaft of one of the long bones. Peyer's patches in the intestine were swollen and ecchymotic. The mesenteric glands were somewhat enlarged. The next case had been that of a man, seventy-two years of age. The first sign had been enlargement of the spleen, and this had steadily increased up to the time of death. Four months after enlargement of the spleen the cervical, submaxillary, and inguinal lymph nodes had enlarged. At the time of coming under observation the



spleen had reached two inches below the umbilicus. The liver reached four inches below the free border of the ribs. There had been no fever, and no heart murmurs. Two or three blood counts had been made. The hæmoglobin had been about sixty-five per cent., and the red cells had never been below 4,500,000. There was over nine per cent. of large mononuclear cells. It had been noted that the cells took up Ehrlich's nuclear stain with great difficulty. Death occurred nine months after the first symptoms, from pneumonia. The next case had been that of a man between fifty and sixty years of age, who had gradually emaciated for nearly a year before his death. The spleen had been very markedly enlarged, and the inguinal, abdominal, and axillary lymph nodes had also been enlarged. The blood showed the ratio of at least one white to five red cells, and of the white cells over ninety per cent. were small lymphocytes. There were no mast cells, myelocytes, or eosinophiles. Death had occurred in a little over one year. The fifth case had been that of a man fifty-seven years of age, who had had the grip three years before, followed by slight enlargement of the cervical and axillary glands. Nineteen months before death the lymph nodes on each side of the neck had begun to increase in size, and at the same time blood clots had been passed in the urine. The latter was explained by an ulcer in the urethra. Several times bacilli resembling tubercle bacilli in appearance had been found in the sediment from the urine, which had not been decolorized by remaining twenty-four hours in alcohol. The enlargement of the neck had been so great that the neck had measured nineteen and one-half inches in circumference. The spleen had reached to three fingers' breadth below the ribs. The liver had been only slightly enlarged. The hæmoglobin was thirty-five per cent.; red cells 2,000,000, and white

cells 418,000. The white cells were over ninety per cent., almost all being of the small variety. The duration of the illness was seven months. The speaker said that the most interesting feature of these cases was that the count of the white cells had apparently run parallel with the duration of the disease, the cases having the highest count of white cells having lasted the longest.

Dr. E. LIBMAN said that he had seen five cases of acute leukæmia, and they had been of a very different type from the chronic ones. The term "acute lymphocythæmia" seemed to him a much better one. In the cases that he had seen, the white cells had consisted for the most part, of mononuclear cells, which ranged from the size of the red corpuscle to twice that size. There was but little protoplasm about the nucleus, and the nucleus stained poorly, and was poor in chromatin. The polynuclears were relatively, and sometimes absolutely, diminished in number, and the eosinophiles were very few. Fränkel had described such cases. The first case that he had seen had been that of a child who had been admitted to the Mount Sinai Hospital after an illness of a few weeks. At that time there had been moderate enlargement of all the lymphatic glands, a very large spleen, and hemorrhages all over the body. He had been impressed at that time with the fact that the red cells showed the characteristic picture of pernicious anæmia. Otherwise the picture was that of an acute leukæmia. Many of the white cells appeared to be degenerating. Two days later the white cells had decreased very markedly. Corresponding to this, the spleen and the lymphatic glands had been reduced almost to the normal. Before death the white cells numbered only 2,200, and the blood-picture was that of a pernicious anæmia. There had been a pneumonia present to which was attributed the regression of



the leukæmic symptoms, as it is known that under the influence of acute infectious processes leukæmic changes are apt to diminish. Another of the cases he had seen had resembled typhoid fever. In a third case there had been two small abscesses, and the contents of these consisted almost entirely of mononuclear cells. The fourth case had been that of a woman who had entered Mount Sinai Hospital suffering from pneumonia. She had the color of pernicious anæmia, and had hemorrhages in the skin and from the gums. The white cells had not been increased greatly, but had been of the type described. It was considered that the patient was suffering from an acute leukæmia which was regressing because of the pneumonia, and an unfavorable prognosis was made. The pneumonia resolved entirely but the patient died some days later. Dr. Libman said that he had reason to believe that the symptoms and blood changes in an acute leukæmia could regress without the presence of an infectious process, and that such regression would have to be explained theoretically by an exhaustion of the bone marrow, due to the hemorrhages.

Dr. JAMES EWING said he could accept three of the cases as rather typical, but the evidence in the first two cases of acute lymphatic leukæmia seemed incomplete. In the first there had been a moderate number of myelocytes and megaloblasts. The anæmia had been severe, and there had been no autopsy. Moreover, the lymphatic enlargement had been very slight. Such evidence seemed to him insufficient to establish the diagnosis. Myelocytes were almost unheard of in lymphatic leukæmia—it was a diagnostic point. The megaloblasts were usually very scarce; from the mild stages of the chronic cases up to the termination of the disease the absence of megaloblasts was quite char-



acteristic. The lymph nodes were usually the chief seat of the lesion, and were very prominent, yet in this case they had been but very slightly affected. While, therefore, he would not say that it was not a case of acute lymphatic leukæmia, there was good ground for hesitating to accept that diagnosis. In the second case, megaloblasts had been present; the spleen had been extremely small, as had also been the intestinal lymph nodes. The absence of any distinct indication of hyperplasia of the lymphatic structures was a strong point against acute lymphatic leukæmia. He understood that there had been a considerable enlargement of the liver during life—even more marked than that of the spleen—while the blood had shown the white cells to be not small lymphocytes, but large mononuclear cells. He would not, therefore, classify the case as one of acute lymphatic leukæmia, but rather as one of Von Jaksch's anæmia. He had seen over one hundred thousand leucocytes in a case of diphtheria in a child, over ninety per cent. of which had been small lymphocytes. He would not make a diagnosis of acute lymphatic leukæmia unless the lymphatic structures were very much enlarged, or the blood changes very distinct, or until he had made a microscopical examination of the bone marrow. It was beginning to be recognized that the separate types of leucocytes had nothing to do with one another. In typical cases of lymphatic leukæmia the small lymphocytes were the ones specially increased, yet on examining the slide presented it seemed to him that the small lymphocytes were hardly increased at all.

Dr. LIBMAN said that from the examination of the blood-slide from the second case, he thought there could be no doubt that it was a case of acute leukæmia. These cases were entirely different from lymphatic leukæmia. The term "acute leukæmia" was introduced to describe

cases in which there existed fever, hemorrhages, enlargement of the spleen and lymphatic glands, involvement of the bone marrow; and Fränkel had shown, in 1895, that the blood changes found were such as appeared in the specimen under the microscope, and that all these changes could disappear to a large extent before death. He had himself seen two cases in which the phenomena had almost entirely disappeared before death, yet the bone marrow presented typical changes at the autopsy. The slide, together with the history, made it clear that the case was one of acute leukæmia, according to the description given by Fränkel.

Dr. JANEWAY, in closing, said that in the first case the spleen had decreased from one-half to three-fourths during the last few days of life. By comparing the size of the mesenteric lymph nodes as found at autopsy with the clinical record it seemed evident that the lymph nodes had diminished in size in proportion to the diminution in the size of the spleen. It had been recently suggested by Minkowski to make three classes of leukæmia, viz.: (1) Ordinary chronic leukæmia, beginning rather acutely, remaining stationary for some time, and having a considerable duration; (2) acute leukæmia, having the primary lesion in the bone marrow, just as in the first class, but associated with enlargement of the spleen and of the lymph nodes and lymphatic apparatus elsewhere, and running a very rapid course, sometimes only a few days, and associated with fever and hemorrhages; and (3) the chronic lymphæmias—the class specially referred to by Dr. Ewing. The classification of Von Jaksch's anæmia had seemed to him specially indefinite. A number of cases had been recorded of transition from Von Jaksch's anæmia to acute leukæmia. There was one case on record of transition from pernicious anæmia to acute leukæmia. In his fifth case,



which Dr. Ewing did not question, the cells had not all been of the small lymphocyte type. The presence of megaloblasts in the acute cases had been thought to go hand in hand with the acute anæmia.

DEMONSTRATION OF SPECIMENS SHOWING THE GROWTH  
OF THE TUBERCLE BACILLUS ON HESSE'S MEDIUM.

Dr. ROBERT J. WILSON said that Hesse's medium was supposed to be peculiarly favorable for the growth of tubercle bacilli, and depended for its efficacy upon a special food-stuff. It was claimed that the tubercle bacilli would grow on this in a few hours, so that their vitality could be proved or disproved very quickly. The medium was also useful in cases in which the growth of the bacilli was not very characteristic. Dr. Wilson said that he had made a series of seventeen plates, and growths had been obtained on nine of these. Trials had been made on blood serum and on ordinary agar-agar, and with the exception of one there had been no growth at the end of forty-eight hours. In the exceptional case there had been a small growth limited to the borders of the cell. He had observed that the growth in the plate seemed to start in some of the material carried on to the plate at the time the culture was made. This led him to think that it was highly probable that the growth always first started in such material, and then finding the medium favorable for further growth was able to develop on it. The material transferred to this plate was necessary for the starting of the growth, in very much the same way as blood was necessary for starting cultures of the influenza bacillus. Hesse thought it probable that the tubercle bacillus divided in its longitudinal axis, which was, of course, contrary to the general belief, yet a study of these plates



which he had prepared afforded some ground for such belief.

*Discussion.*

Dr. HISS said that he had had experience in only four cases in the growth of bacilli from sputum on Hesse's medium. In all of these he had obtained a growth which had continued for from twelve to fourteen days after which the colonies had developed very slowly. One plate had been under observation for two months. He had seen nothing to make him think that longitudinal division occurred. The organisms were pressed out of the advancing line along the line of least resistance. It might be that material carried over had something to do with the starting of the growth. The medium might be of service in the rapid differential diagnosis of organisms presenting the same staining characteristics.

Dr. L. A. CONNER asked whether the tubercle bacilli grew very much more rapidly on Hesse's medium than the other bacilli found in the sputum.

Dr. HISS replied that the contaminating organisms from the sputum did not seem to grow so well on Hesse's medium, as did the tubercle bacilli. This was one of the chief advantages of the medium.

Dr. WILSON explained that his work had been done with pure cultures from guinea-pigs, so that he had had no contaminating organisms.

Dr. HISS said that the specimens that he had had under observation for two months had not been over-run in this way, and in the others the colonies were still discrete. Hesse had taken great pains to secure bacilli from material as free as possible from contamination.

Dr. HARLOW BROOKS said that he had seen four plates by Dr. Hesse from sputum which contained a good many streptococci and staphylococci. The contaminat-

ing organisms had been found comparatively common in the smear, while the tubercle bacilli had not been very numerous. About forty-eight hours afterward the colonies of tubercle bacilli were strikingly evident, while the growth of the contaminating colonies had not been very marked. Two or three days later the contaminating colonies had overrun the tubercle growths.

#### A CASE OF INTESTINAL TUBERCULOSIS IN A CHILD.

Dr. D. BOVAIRD reported the case of a boy aged three years, who had been returned to the New York Foundling Hospital last August in accordance with the rules of that institution. He had then been sent to the home at Spuyten Duyvil and had remained in fair health until October 24th. At this time, during an epidemic of measles, he had developed this disease, and it had run the usual course. For a whole month the temperature had been very irregular, and the symptoms had indicated a severe bronchitis or broncho-pneumonia, although the physical examination failed to show pulmonary consolidation. During this time there had been a severe stomatitis, though this had improved slowly. At the end of November the evening temperature had been 100° to 101° F. After about two weeks more of this irregular fever the child had developed the first enteric symptoms. About December 15th the condition of the mouth had again become worse, and had soon changed into a true cancrum oris. It was in this condition that the child had been returned to the Foundling Hospital. Death had occurred from the exhaustion incident to this. On December 20th the child had died, and the autopsy had been made on the following day. The pharynx, trachea, and bronchi were normal. Both pleural cavities, except anteriorly, had been obliterated



by adhesions. The bronchial nodes contained a few minute caseous foci. The lungs themselves were normal. The peritoneal cavity had been obliterated by adhesions, and the intestine and all the other viscera were matted together by adhesions. The peritoneum was thickly studded with miliary tubercles. The peritoneal surface of the liver was covered with miliary tubercles, but only a few were found in the hepatic tissue. The same condition was found in the spleen. The kidneys, suprarenals, pancreas, and stomach were normal. Peyer's patches were swollen and contained numerous ulcers, which reached to the peritoneal coat. In the upper part of the large intestine were a few caseous nodules. The solitary follicles were enlarged throughout and somewhat ulcerated. The bladder was normal except for slight congestion. The mesenteric nodes were enlarged, and some of them showed caseous foci. Among the records of the Foundling Hospital were those of two hundred autopsies on cases of tuberculosis in children, yet in only three was there a fair presumption that the intestine had been the primary seat of the infection. For this reason the case just presented was of special interest. Furthermore, when tuberculosis had developed immediately after measles he had usually assumed that it was nothing more than a lighting up of a previously received tuberculous infection, but in the present instance the diffusion of the lesions through the small intestine, taken in conjunction with the history of the case, made it not improbable that the tuberculous infection had developed subsequently to the measles.

#### *Discussion.*

Dr. EWING thought the development of tuberculosis after measles was too common to suppose that the tu-



berculous infection always occurred after the measles. Regarding the condition of the Peyer's patches it should be borne in mind that these were really excretory organs, and might possibly, under certain circumstances, suffer from the excretion of tubercle bacilli. He had recently seen for the first time cases of intestinal tuberculosis limited entirely to Peyer's patches. He had formerly supposed that tuberculous ulcers were almost invariably irregular, and that their long axes were in the transverse diameter of the intestine. In some recent cases the ulcers had been confined to Peyer's patches, which had been deeply excavated. It seemed possible that the bacilli might sometimes be deposited here by the blood current or by the lymph current, instead of from the intestinal contents.

Dr. BOVAIRD said that the child had been fed on milk obtained from an excellent source. Up to the very last the child had had a diarrhoea, yet the stools had never contained blood, only mucus and undigested food.

Dr. MARTHA WOLLSTEIN said that autopsies had been held at the Babies' Hospital on some hundreds of cases. Probably fifteen per cent. had been on tuberculous infants, yet in few of these children during life had bloody stools been present, so that they had come to look upon this as a rare symptom in young infants. At that hospital they had never met with a single case in which it seemed at all clear that the infection had been primary in the intestine, even in infants fed on bad milk. In the case under discussion it seemed impossible to prove that the tuberculosis had not been primary in the bronchial nodes. Its rapid extension along the intestinal tract was possibly due to the condition of that tract (a previous catarrhal condition of the intestine), thus furnishing a line of least resistance, and explaining the localization.

## A CASE OF ACUTE TUBERCULOUS BRONCHO-PNEUMONIA.

Dr. E. HODENPYL presented specimens from a case of acute tuberculous broncho-pneumonia in which death had occurred on the twelfth day of the disease. The symptoms and physical signs had resembled very closely those of acute lobar pneumonia. The gross lesions of the lung could hardly be distinguished from those of a simple case of broncho-pneumonia. The short duration of the disease was very remarkable. The shortest recorded case of pulmonary tuberculosis of which the speaker was aware was eleven days. The subject was a colored woman, who had been taken sick with pain in the left chest, cough, prostration, and fever. After remaining in bed five days she had been removed to the hospital, and examination had shown apparently an ordinary pneumonia involving the whole lung. The temperature fell rapidly in two or three days from  $105^{\circ}$  to  $96^{\circ}$  F., and in a few hours rose again to  $105^{\circ}$ . At this time evidence of consolidation had been discovered on the opposite side. She died on the twelfth day. At the autopsy, the left lung, which was free from pleurisy, was studded with larger and smaller pneumonic patches arranged about the bronchi. These were so numerous as to cause the lung to appear almost solid. The pneumonic patches except in one or two instances, failed to show any cheesy degeneration. The right lung presented the same lesions as the left, though developed to a less degree. Stained smears from the pneumonic exudate showed tubercle bacilli in enormous numbers.

*Discussion.*

Dr. PRUDDEN asked if there was any clew as to the possible source of the infection.

Dr. HODENPYL replied that he could find none. Evi-



dently this was a case of aspiration tuberculosis from the bronchi. No old tuberculous focus had been made out.

---

*Stated Meeting, February 14, 1900.*

EUGENE HODENPYL, M.D., PRESIDENT.

A CASE OF MALARIA FOLLOWING WOUND INFECTION.

Dr. F. M. JEFFRIES reported this case. A surgeon of this city who had never had malaria, and who had been in the city up to July 15, 1899, had then gone to Seabright, and had remained there from July 15th to 20th. While there he had driven around during the daytime, but had remained indoors at night. This neighborhood was not considered a particularly malarious one. From July 25th to September 15th he had been in the northern part of New Hampshire, where malaria is entirely unknown. He had returned to this city on September 15th, and on September 18th had performed a vaginal hysterectomy on a patient who, the next day, had had a distinct and marked malarial chill. This patient was from North Carolina, and had previously had frequent attacks of malaria. The next two days she had had chills and as she had been given quinine on the second day there had been no further chills. At the time of the chill examination had shown no evidence of infection. During the operation referred to the surgeon had pricked his finger several times. On October 4th, or sixteen days after the operation, the surgeon had had his first chill. This had been followed by several others, each attack lasting for four or five days. On November 24th, or just prior to one of the paroxysms, the speaker had examined his blood and had found the plasmodium malariae in great abundance. It was of the variety known as the æstivo-autumnal—a variety not commonly



met with in this locality except in those who have had it previous to coming here. The evidence presented by this history certainly led one to at least suspect very strongly that the surgeon had become infected with malaria by wound infection. Specimens of the blood were exhibited under the microscope.

#### A CASE OF ADVANCED TUBERCULOUS COCCYTIS.

Dr. JEFFRIES showed an extreme case of tuberculosis of the hip. The specimen had been removed from a girl, aged ten years. The acetabulum was almost wanting, and the head and neck of the femur were entirely gone. Pus had burrowed as far back as the sacrum. The right hip was normal. In the left knee joint was found a nail such as is used in holding together the bones after excision of this joint, but there was no history of such an operation having been done in the hospital in which she had died. At the autopsy the upper right apex of the lung contained a small tuberculous nodule. The liver and spleen were enlarged and amyloid. The bladder showed a few tuberculous ulcers. The left kidney was fatty and tuberculous in its upper half. The uterus presented a tuberculous endometritis, and there was also a tuberculous salpingitis. The mesenteric glands were enlarged throughout.

#### *Discussion.*

Dr. CARLIN PHILLIPS asked whether the woman upon whom the operation of vaginal hysterectomy had been done had originally had the æstivo-autumnal variety of malaria.

Dr. JEFFRIES replied that this patient had come from a region in which this variety was common, but there had been no opportunity of determining this point positively.

## TWO CASES OF PERFORATION OF TUBERCULOUS BRONCHIAL LYMPH NODES INTO THE TRACHEA.

Dr. F. S. MATHEWS presented the first of these cases. The specimens had been taken from a child, aged four years, who had been in the St. Mary's Hospital for Children for several months under treatment for tuberculous disease of the knee. On the night of January 19th the child, while apparently in his usual health, had suddenly begun to cough, and had soon choked to death. At the autopsy, made the next day, there had been found on the right side an opening from which pus was oozing. This connected with the abscess cavity. The trachea and right bronchus contained a considerable quantity of pus. Tubercle bacilli were found in this pus, and also in the mediastinal glands. No other tuberculous lesions had been found in the thoracic or abdominal cavities.

Dr. M. NICOLL, JR., presented the history of a second case. The specimens had been taken from a child one year and a half old, who had been in the out-patient department of the New York Foundling Hospital under treatment for bronchitis for about one week. The child had then been taken into the hospital because of the urgent dyspnoea and cyanosis present. Expiration was more difficult than inspiration. The child had been immediately intubated, but without relief, and death had taken place in a few minutes. On opening the thorax the left lung had been found over-inflated, and in a condition of very acute emphysema. On cutting through the left primary bronchus a large plug of mucus had forcibly escaped, and the lungs had then immediately collapsed. In the trachea, near the origin of the right primary bronchus, was an oval opening communicating with a cavity formed by the breaking down of a large

tuberculous gland. The bronchus leading to the right lower lobe ended in a mass of tuberculous consolidation. The over-distention of the left lung was apparently due to the valve-like action of the inspired mucus plug; the power of expiration was not sufficiently strong to expel the air taken in at each inspiration.

A CASE OF MEMBRANOUS LARYNGITIS COMPLICATING  
TYPHOID FEVER.

Dr. ALEXANDER LAMBERT presented the larynx from a person who had died of gangrenous laryngitis. The man was an alcoholic subject who had entered the hospital with a temperature of 105° F., marked dyspnœa, severe cough, and laryngitis. The leucocytes had been reported as being only five thousand, and on that basis a diagnosis of typhoid fever had been made. The next day the spleen had become palpable, and a profuse typhoid eruption had appeared. Toward the end of the third week the temperature had fallen very decidedly, and the man had shown every evidence of improvement. About this time the laryngitis had become much more severe. The fauces were red, and in the back of the pharynx was a slight grayish deposit. In the course of three days aphonia had become complete. Preparations had been made for a rapid tracheotomy should suffocation seem imminent. A tracheotomy had finally been demanded for an acute attack of suffocation, and this had given great relief. At midnight, however, the man had died suddenly of heart failure. At the autopsy a thick membrane was found extending from the epiglottis down into the larynx, and in the sulcus on the left side was an abscess cavity. There was also a peribronchitic inflammation which would probably have resulted in time in a septic broncho-pneumonia. There were some ulcers in the



cæcum, which were practically healed. On searching the literature he had found that Keen had reported most of the cases, and that most of them had developed well on in the period of convalescence. Often the first symptom had been aphonia, or evidence of suffocation, and death had often occurred almost instantly. In two of the reported cases the patients had recovered without tracheotomy, and two with it. The gangrenous area extended from the epiglottis down two and a half inches into the trachea itself.

*Discussion.*

Dr. E. K. DUNHAM said that he had examined microscopically the specimens from this case. The most interesting of these had been the one taken from the pharyngeal surface of the aryteno-epiglottic fold. In this there had been a diphtheritic inflammation, and a line of demarcation had formed, causing a loosening of the necrotic tissue. A little beneath this line of demarcation there was an acute exudative inflammation with interstitial hemorrhage. Apparently, therefore, there had been two infections; an earlier one causing necrosis of the surface, and a later one causing an interstitial hemorrhage at the base of the granulation tissue. On the inner or laryngeal aspect of the aryteno-epiglottic fold there was a fibrinous exudate on the surface with granulations beneath, and a rather excessive diapedesis of red corpuscles. This exudate was probably due to the second infection. Over the cricoid the condition was very much the same as on the inner surface of the aryteno-epiglottic fold. Nearly all the microorganisms found were diplococci or streptococci.

Dr. GEORGE P. BIGGS asked why this case was described as a gangrenous laryngitis.

Dr. LAMBERT replied that the tissue in the upper and

back part of the pharynx had been perfectly black and gangrenous.

A CASE OF ANEURISM OF THE AORTA.

Dr. ALEXANDER LAMBERT reported this case. The subject was a man, alcoholic, forty years of age. Syphilis had been suspected, but not definitely made out. He had had an attack of pain in the shoulder with dyspnoea some years before. Five years before that attack the man had walked into the Vanderbilt Clinic with typhoid fever, and at that time the aneurism had been discovered. During 1898 and 1899 the man had worked laboriously and drunk excessively. When seen last summer, there had been a large mass projecting from under the chin. This mass measured vertically five and a half inches. Physical examination had revealed evidence of compression of the left lung. The man had left the hospital in August. When seen again, on November 14th, the tumor had extended from the deltoid muscle on the left side across to within three inches of the opposite deltoid. It measured eleven and a half inches transversely and projected three and a half inches. Its circumference was twenty-four inches. At that time the skin had been exceedingly thin over the tumor. About a month later the tumor had measured thirteen inches transversely. Five days after this the man had coughed up a rather large clot of blood. One or two nights afterward the aneurism had ruptured. The autopsy showed that the anterior wall of the aneurism was made up of the skin, and that there was no true sac. The entire thoracic wall had been eroded by the tumor. The blood clot weighed 1500 gm. The cartilages on the left side had been completely absorbed, and the ends of the ribs had been eaten away. The lungs, trachea, and larger

bronchi were normal. There was hypertrophy of the left ventricle of the heart, but the valves were normal. The lateral portion of the arch opened into the aneurism by a large aperture. The interesting feature about the aneurism was that a hole had been made in the aorta by the giving way of an atheromatous patch. The blood had then been diffused into the areolar tissue of the mediastinum, and had become organized there. This mixture of connective tissue and coagulated blood had formed the aneurismal sac.

*Discussion.*

Dr. BIGGS took issue with Dr. Lambert as to the explanation of the formation of the aneurism. He thought there had probably been the usual gradual development due to local weakening of the vessel rather than to a rupture of the vessel.

Dr. LAMBERT replied that it was probable that the aneurism had started originally like other aneurisms, but there was, at the time of the examination, no longer any evidence of the first formation.

TWO CASES OF ACUTE INTESTINAL OBSTRUCTION FROM  
INTUSSUSCEPTION.

Dr. H. J. BOLDT presented specimens from two cases of acute intestinal obstruction due to intussusception. In the first case the intussusception had been caused by a polypus. The second specimen was from an intussusception occurring in a child as a result of a diverticulum in the bowel. He said that he had never met with a case of this kind in which cathartics had not been administered, and he wished to direct attention to the great importance of avoiding the administration of such drugs. If an acute intestinal obstruction could



not be relieved in a few hours by the use of high enemata with inversion of the patient, and massage of the intestines, an abdominal section should be performed ere serious symptoms became manifest.

#### GANGRENOUS DERMOID FROM TWISTING OF THE PEDICLE.

Dr. BOLDT also exhibited a gangrenous dermoid tumor of the left ovary. The condition had been produced by a complete twist of the pedicle from left to right. Apparently the condition had lasted for three or four days.

#### A CASE OF SUPPURATIVE METRITIS.

Dr. GEORGE P. BIGGS presented specimens taken from a woman, thirty years of age, who gave birth to a full-term child sixty-three days before death. On the seventh day after confinement she had a chill, followed by fever and pelvic pain. Curettage was done two days later and the symptoms gradually abated so that three weeks later she was able to sit up. After two days she was obliged to return to bed for three weeks, but was up again for two weeks before coming to the hospital. She entered the New York Hospital sixty-one days after parturition, and at that time was very anæmic and markedly septic. On examination considerable bloody fluid escaped from the uterus, and a sound passed directly into the peritoneal cavity. Her condition was so bad that a radical operation could not be attempted, but free drainage was established through the uterus and posterior cul-de-sac. At the autopsy marked pelvic peritonitis with extensive adhesions was found. The uterus was but little larger than normal and showed an area of advanced softening 2 cm. in diameter in the anterior portion of the fundus. It was through this area that the sound had passed into the peritoneal cav-

ity. The right corner of the uterus was 3 cm. in diameter and contained a series of communicating abscesses. In the left broad ligament there was an abscess 3 cm. in diameter. Smears and cultures from these abscesses showed an abundance of streptococci. The Fallopian tubes and ovaries on each side appeared normal except for surface inflammation. Interesting points in the case were the distinct suppurative metritis which the speaker had not seen before, and the long duration for such a process of necrotic softening. The cæcum from this case was much contracted, and its wall was thickened and ulcerated. The process was evidently one of long standing. Partly from a few old tuberculous lesions in the lungs, and partly from the presence of a few miliary tubercles in the peritoneum over the site of a similar ulcer in the ileum, he inferred that this was a tuberculous process.

#### *Discussion.*

Dr. BOLDT commented upon the small size of the uterus, in view of the amount of sepsis present. Furthermore, the uterus was much firmer in consistency than in a case of puerperal sepsis. The suppurative metritis present in this case was exceedingly interesting. He had seen only one other case somewhat resembling this.

Dr. AUGUST JEROME LARTIGAU then read a paper on

#### TYPHOID INFECTION OF THE UTERUS.

As a natural sequence and derivative of the study of the experimental side of typhoid infections, and coexistent with the evolution of the conception of typhoid fever, may be traced the development of our knowledge of the ubiquity of the typhoid bacillus in human pathology. Appreciation of its rôle in various pathological processes finds ample expression in the abundance of

literature upon this subject, largely as examples of post-typhoid lesions. But notwithstanding the rapidly augmenting number of reports of atypical typhoid infections, instances of invasion of the uterine cavity by this micro-organism are limited to a few recorded examples published within comparatively recent date. The great interest recently manifested in the pyogenic attributes of the typhoid bacillus and the rarity of its localization in the uterus have led me to report the two cases which form the chief subject of this paper.

Regarded as an anticipation of the subsequent bacteriological realization of this form of disease, Kühnau's<sup>1</sup> case is not without interest. The patient was a woman, aged thirty-two years, who was admitted to the hospital on April 29, 1895. For some time previous she had been under observation in the outdoor department. She was taken sick on April 20th, and labor set in April 26th; the delivery was normal, and the child was born alive and healthy. The day preceding entrance into the hospital there were somnolence and delirium. On admission, the temperature was 39° C., the abdomen was distended, and some ileo-cæcal gurgling was noted. The spleen was not palpable, and the liver apparently not enlarged; there was some diarrhoea. An examination of the urine showed the presence of some albumin and the diazo reaction. On May 6th a few rose spots were detected; the spleen was just palpable. The following day several foul-smelling clots were removed from the uterus. Three days later a culture taken from the blood contained typhoid bacilli. Finally, on June 17th, the patient died. At the autopsy no intestinal lesions were observed; the mesenteric glands were swollen, and showed areas of necroses and

<sup>1</sup> Kühnau: Ein Fall von Septicopyæmia typhosa. *Berlin. klin. Wochenschr.*, July, 1896, p. 667.



abscesses; the kidneys also contained abscesses, and the left ovarian vein contained a partially softened thrombus. The cervix was covered with a hemorrhagic membrane.

The bacteriological examination of the pus from the mesenteric lymph glands, kidney abscesses, thrombus in the ovarian vein, and splenic pulp demonstrated bacilli morphologically similar to and culturally behaving like the bacillus of typhoid fever. In the discussion of his case Kühnau argues two possible sources of infection—one from the uterine cavity and the other from the intestine. For neither has he any positive evidence in the form of bacteriological support.

In contradistinction to this observation may be cited the more conclusive examples studied by G. W. Dobbin<sup>1</sup> and George Blumer,<sup>2</sup> who were able to demonstrate the presence of the bacillus of typhoid fever in the cultures taken in one instance from the lochia and in the other from the uterine cavity proper. Both cases occurred in puerperal women, unlike our own in that respect.

The case of Dobbin was in a woman, aged twenty-four years, who was admitted to the Johns Hopkins Hospital on November 17, 1897, complaining of fever following confinement. Five days before she had given birth to a child, that lived a day and a half. Up to the time of her confinement she had been well. On the second day post-partum, November 13th, she was found to be suffering from bronchitis, with a temperature of 103° F. The following day several clots and pieces

<sup>1</sup> George W. Dobbin. A Case of Puerperal Infection in which the *Bacillus typhosus* was found in the Uterus. *American Journal of Obstetrics*, August, 1898, p. 185.

<sup>2</sup> George Blumer. A Case of Mixed Puerperal and Typhoid Infection in which the Streptococcus and the Typhoid Bacillus were isolated both from the Blood and the Uterine Cavity. *American Journal of Obstetrics*, 1899, vol. xxxix., No. 1.

of placenta were removed from the uterus. When admitted to the hospital her temperature was  $103.1^{\circ}$  F., and the physical examination revealed a papular eruption on the lower thorax and abdomen, which in places simulated rose spots. There was no enlargement of the spleen, but some iliac gurgling and tympanites were present. The uterus was large and movable. The fever was irregular, running as high as  $107^{\circ}$  F. for the first week after admission, but it gradually declined from this time. Blood obtained on the fifth day gave a well-marked positive Widal reaction; cultures from the median basilic vein remained sterile. The bacteriological examination of the uterine lochia showed the presence of the *Streptococcus pyogenes*, the *Staphylococcus pyogenes aureus*, typhoid bacillus, and an unidentified bacillus. Dobbin thinks that the source of infection was from without. I am constrained to regard this, however, as extremely improbable.

More recently Blumer published his case, which was that of a married woman who was confined by a midwife. On the sixth day of the puerperium the patient, shortly after a hearty meal, was taken with dyspnœa and incoherency of speech, and rapidly became delirious and semi-comatose. When seen by a physician, about four hours after the appearance of her trouble, the temperature was  $100.8^{\circ}$  F., pulse 120; the tongue was almost black and swollen to two or three times its normal size. The heart and lungs seemed normal. The abdomen was greatly enlarged, but nowhere tender; the liver dulness reached one third of the way down from the costal margin to the umbilicus. The spleen dulness was much increased. The uterus was as much contracted as could normally be the case thus soon after labor. The urine was constantly passed involuntarily. Bowels constipated. Scattered over the chest and abdomen was a fine, papular rash, in places suggestive of



rose spots. Two days after the onset of her sickness the patient died. The child remained alive and showed no signs of any illness resembling typhoid fever. At the autopsy there were found swelling and ulceration of Peyer's patches in the lower end of the ileum, acute spleen tumor, swelling of the mesenteric lymph glands, subinvolution of the uterus with partial retention of the placenta, multiple gas cysts in the small intestine, and emphysema of the subcutaneous tissues of the neck and back. The cultures from the heart's blood, liver, spleen, and uterus contained the bacillus of typhoid fever and the *Streptococcus pyogenes*. It is also probable that the *Bacillus aërogenes capsulatus* was associated with these micro-organisms. The *Bacillus typhosus* existed alone in the mesenteric glands.

The problem of bacterial transmission from mother to foetus is so inseparably connected in some of its phases with the question of uterine infection in this group of cases that explanation, at all events a part of it, will necessarily be sought for in the results of experimentation and observations along these lines. That bacteria filter through the placenta into the blood of the foetus, with or without demonstrable placental lesions, has received final proof. Although the passage of micro-organisms from the mother to the foetus has been abundantly proved by the researches of Malvoz,<sup>1</sup> Wolff,<sup>2</sup> Birch-Hirschfeld,<sup>3</sup> Simon,<sup>4</sup> Lubarsch,<sup>5</sup> and others, it cannot

<sup>1</sup> Malvoz. Sur la transmission intra placentaire des micro organismes. *Annales de l'Institut Pasteur*, 1888, p. 121.

<sup>2</sup> Wolff. Ueber Vererbung von Infektionskrankheiten. *Virchow's Archiv*, Bd. cxii., p. 196.

<sup>3</sup> Birch-Hirschfeld. Ueber die Pforten der placentären Infection des Foetus. *Ziegler's Beiträge zur pathol. Anatomie*, etc., Bd. ix.

<sup>4</sup> Simon. Beiträge zur Lehre von dem Uebergang pathogener Micro-organismen von Mütter auf Frucht. *Zeitschr. für Geb. und Gyn.*, Bd. xvi., Heft 1. Speier's Inaugural-Dissertation.

<sup>5</sup> Lubarsch. Uebertragung von Infektionskrankheiten von Ascendenten auf Descendenten. Lubarsch und Ostertag, *Ergebnisse*, 1896, Bd. i.



be said that the ultimate conditions underlying its occurrence have been shown. Chantemesse and Widal,<sup>1</sup> Hildebrand,<sup>2</sup> Eberth,<sup>3</sup> Ernst,<sup>4</sup> Frascani,<sup>5</sup> Janiszewski,<sup>6</sup> Freund and Levy,<sup>7</sup> Dürck,<sup>8</sup> Étienne,<sup>9</sup> and Speier<sup>10</sup> have contributed a small number of observations of the transmission of the typhoid bacillus from mother to foetus in human beings—suggestive facts which lend additional weight to the internal origin of this type of infection, certainly for the two cases reported by Dobbin and Blumer. The presence of typhoid bacilli in the intestinal dejecta and their frequent occurrence in the urine of patients already suffering from typhoid fever will probably be shown to be important considerations in the determination of the mode of invasion in another still rarer category of cases.

For the clinical histories of the following cases I am indebted to Dr. Howard Van Rensselaer and Dr. Willis G. MacDonald, of Albany, New York.

<sup>1</sup>Chantemesse and Widal. *Bacille typhique et étiologie de la fièvre typhoïde. Archives de physiologie*, 1887, p. 19.

<sup>2</sup>Hildebrand, G. Zur Casuistik des placentären Ueberganges der Typhusbacillen von Mutter auf Frucht. *Forts. d. Med.*, 1889, Bd. vii., No. 23.

<sup>3</sup>Eberth, C. J. Geht der Typhusorganismus auf den Foetus über? *Fortschritte der Medicin*, 1889, Bd. vii., No. 5.

<sup>4</sup>Ernst, P. Intrauterine Typhusinfektion einer lebensfähigen Frucht. *Ziegler's Beiträge zur path. Anat.*, etc., Bd. viii.

<sup>5</sup>Frascani. Osservazioni cliniche e ricerche sperimentali sul passaggio del bacillo del tifo dalla madre al feto. *Rivista generale ital. di clinica med.*, 1892, pp. 282, 348.

<sup>6</sup>Janiszewski, Th. Uebertragung des Typhus auf den Foetus. *Münch. med. Wochenschrift*, 1893, No. 38.

<sup>7</sup>Freund und Levy. Ueber intrauterine Infection mit Typhus abdominalis. *Berliner klinische Wochenschrift*, 1895, No. 25.

<sup>8</sup>Dürck. Ueber intrauterine Infection mit Typhus abdominalis. *Berliner klinische Wochenschrift*, 1895, No. 25.

<sup>9</sup>Étienne. *Gaz hebdom. de méd. et de chirurg.*, 1896, No. 16.

<sup>10</sup>Speier. Zur Casuistik des placentären Ueberganges der Typhusbacillen von der Mutter auf die Frucht. *Inaugural Dissertation*, Breslau 1897.

CASE I.<sup>1</sup>—Mary L. J., married, aged thirty-one years, American, entered the Albany Hospital, March 11, 1899, complaining of general abdominal pain and tenderness, most marked in the region of the left iliac fossa.

*Family History.*—Father died of tuberculosis. Mother alive and well. Two brothers died of meningitis in infancy. One brother and four sisters alive and well.

*Personal History.*—As a child she suffered from measles, chicken-pox, and mumps. During the month of November, 1898, the patient was sick for three weeks with an acute febrile malady, which was diagnosticated typhoid fever.

*Present Illness.*—This began about five weeks ago, when she was taken ill with sharp, paroxysmal pains, starting behind in the lumbar region and extending forward on either side toward the umbilicus. For the first two weeks the attacks were infrequent, but since have progressively increased both in frequency and severity—so much so that at the time of admission the pain is constant, and referred in large part to the iliac region on the left side. The last menstruation occurred in the first week in January, 1899.

On entrance into the hospital the temperature taken in the mouth was 102.4° F.; pulse 118, regular, of fair volume, and not dicrotic; respirations, 27. The build is slim, body moderately well nourished; intelligence good. Pupils are normal and react to light; conjunctivæ normal. Tongue is covered on the dorsum with a thin white fur. No œdema. No eruption on the skin. Examination of the heart is negative. The lungs give no evidence of any abnormality. Spleen is not palpable; the liver is apparently normal. The abdomen is tender, more particularly on the left side in the iliac fossa.

<sup>1</sup> This case has already been reported in connection with another subject. *New York Medical Journal*, July 29, 1899.



Palpation is negative. Vaginal examination shows a normal cervix. Bimanual palpation: Uterus about normal in size, the body directed forward, and freely movable. On the left side an indefinite mass the size of a hen's egg, not freely movable, and firm to the touch. The examination of the opposite side is negative.

*March 12th.*—An abdominal section was done at 12.25 P. M. Diagnosis of ectopic pregnancy confirmed, and removal of left tube and ovary with pregnancy. The patient returned in good condition; pulse, 125; respiration, 30. At 6 P. M., complained of pain in abdomen; pulse, 115 and intermittent; temperature, 101° F. Urine normal.

*13th.*—Considerable nausea and vomiting; evening temperature, 100.6° F.; less pain in abdomen.

*14th.*—Nausea and vomiting persist. Complains of headache; restless; temperature, 100.1° F.; pulse regular, of good volume; heart sounds normal. Lungs showed nothing abnormal. Slight blood-stained discharge from uterus.

*15th.*—Dyspnoea during the night; mind clear; respirations, 36; pulse, small volume, intermittent, 150 to the minute; temperature at 8 A. M., 100.2° F.; at 1 P. M., 102.6° F. Died at 2 P. M.

*Autopsy* (an hour and a half after death).—Anatomical diagnosis: Typhoid fever without intestinal lesions; laparotomy wound (removal of left tube and ovary for extra-uterine pregnancy); acute splenic tumor; cloudy swelling of the liver and kidneys; endometritis; triple-phosphate calculus in pelvis of right kidney.

Body a hundred and sixty-one centimetres long, of slim build and sparsely nourished. Surface of the body generally pale. Rigor mortis absent in upper and lower extremities. Slight livor mortis of the dependent parts. Pupils midwide and equal. Mucous membrane pale.



In the midline of the abdomen is a linear incision eleven centimetres long, extending from the pubis upward, which appears perfectly healthy.

Brain and cord not examined. Abdomen: Parietal peritoneum presents a normal appearance; the visceral layer is smooth, glossy, and moderately injected, especially that portion over the lower third of the ileum. Omentum contains a few discrete pin-head to pea-sized hemorrhages. Foramen of Winslow not patent; diaphragm on the right side, fourth space; on the left side, fifth space, in the mammary line. Thorax: Both pleural cavities are free from fluid. The pericardium is normal; the heart is distended with fluid blood and some red clots and shows no abnormality. Both lungs are free from adhesions, crepitant, and on section light pink in color. Bronchi and pulmonary blood-vessels normal. The spleen is adherent to the omentum by a few old bands; the organ measures  $15 \times 9 \times 5.5$  centimetres; the capsule is smooth and not wrinkled; consistence diminished; the cut surface reddish-brown; the trabeculæ are normal and the pulp is apparently augmented; Malpighian bodies are prominent. Liver: Increased in size; capsule smooth; consistence softer than normal; on section, of an opaque, grayish color and the lobules indistinct. Gall bladder: Moderately distended with thick, dark colored bile. Kidneys: Slightly swollen, especially the cortical portion of the organ; consistence about normal. The pancreas, suprarenal capsules, and retroperitoneal glands show nothing abnormal. The same may be said of the aorta, stomach, bladder, and vagina. The mesenteric lymph glands are not swollen. The intestine shows absolutely no evidence of past or recent changes; the mucous membrane is normal in appearance. The uterus measures  $5.5 \times 4 \times 3$  centimetres; the consistency is normal; the peritoneal surface

is smooth, glossy, free from adhesions, and moderately injected. The walls are not increased in thickness; cavity patent; endometrium soft, red, congested, markedly so in fundus of the organ. Covering the mucosa is a considerable quantity of thick, sticky, hemorrhagic mucoid material.

*Microscopical Examination.*—The tissues studied were all hardened in ninety-five per cent. alcohol. The sections of hardened tissue were stained with hæmatoxylin and eosin, Van Gieson's picro-acid-fuchsine, and by Flexner's methylene-blue and Gram-Weigert methods.

Histological examination of the heart shows nothing beyond a slight degree of fragmentatio myocardii, with dilatation of the capillaries and small veins, in both of which blood may be seen showing a leucocytosis of polynuclear forms. The sections from the lung, intestines, pancreas, and adrenals add nothing to the macroscopical findings. In addition to well-marked cloudy swelling and some more or less localized areas of fatty degeneration, the liver shows no other changes. The "lymphomata" commonly found in the liver of ordinary cases of typhoid fever were entirely absent, as were also any areas of focal necroses. In the sections of the spleen some hyperplasia of the lymph cells was noted, with considerable congestion. In the kidney the epithelium of the convoluted tubules was markedly swollen, granular, and often devoid of nuclei; some of the epithelial cells were desquamated. Uterus: The superficial strata of epithelial cells were desquamated; those remaining, swollen; submucous and mucous tissues filled with polynuclear leucocytes, small round cells, *débris*, and partly broken-down cellular elements. Musculature normal.

Sections of the kidney and lung stained with Weigert's fibrin stain showed very few diplococci, limited to

the small blood-vessels. In sections of the liver, ileum, and uterus, stained by Flexner's method, occasional bacilli (a few clumps in the liver), morphologically similar to the bacillus typhosus, could be distinguished; in the sections of the uterus the bacilli were only made out in the mucosa. Cocci were likewise detected in the mucosa of this organ.

*Bacteriological Examination.*—Agar and gelatin plate cultures were made from the blood of the heart, left lung, spleen, liver, gall bladder, kidney, operation wound, peritoneal cavity, and uterine cavity. The cultures from the lung, operation wound, and peritoneal cavity remained sterile. From the heart's blood and spleen a pure growth of an oval or elongated coccus, sometimes in pairs, more often single, was isolated, corresponding in its tinctorial and cultural reactions to the *Diplococcus lanceolatus*. It was pathogenic for rabbits, both in subcutaneous and intravenous inoculations.

The plate cultures from the uterus contained two sets of colonies—about twenty-five to thirty small pinpoint, gray, translucent, discrete colonies in the depths of the medium. Under the low power they appeared finely granular, oval in form, with a regular outline; the second set consisted of seventy or eighty discrete pin-head sized or slightly larger white colonies, which by transmitted light had a yellow tinge. The latter were made up of not very long, rather thick bacilli, while the former consisted of round cocci, sometimes single, grouped, or in short chains. Culturally, the colonies made up of bacilli have behaved as follows:

Slants of agar-agar: A profuse, slightly elevated, moist, white growth with regular margins.

Blood serum: The growth was equally rapid with that on agar-agar, and presented a similar appearance..

Gelatin stab: A fine, somewhat scanty, white growth



along the line of inoculation. No liquefaction of the gelatin at the end of sixteen days.

Potato: A moist, glistening, scanty growth, which was just perceptible to the eye.

Litmus milk: Very slight acidification of the medium, but no coagulation of the milk.

Alkaline bouillon: The medium became uniformly cloudy at the end of twenty-four hours.

Dunham's peptone solution: The appearances were identical with those of the bouillon. No indol reaction could be obtained.

Stab inoculations into glucose, lactose, and saccharose agar showed a white growth along the line of inoculation, but gave no evidence of gas formation.

Hanging-drop preparations from twenty-four-hour-old bouillon cultures contained bacilli which were actively motile. In cover slips from similar young cultures stained by Pitfield's method might be seen bacilli having ten to twelve flagella (peritrichal arrangement). Tested with sera from known cases of typhoid fever, positive Widal reactions were obtained. Blood taken from the subject post-mortem gave a positive Widal reaction with this bacillus as well as with other known cultures of the bacillus of typhoid fever.

Bacteriological diagnosis: *Baccillus typhosus* (Eberth-Gaffky). Pure growths of the typhoid bacillus culturally and morphologically similar to the above were isolated from the liver, gall bladder, and kidney. The coccus associated in the uterus with the typhoid organism was identified as the *Streptococcus pyogenes*. One cubic centimetre of a young bouillon culture of the streptococcus subcutaneously injected into a three-quarter-grown rabbit produced no untoward effects beyond a moderate local reaction.

CASE II.—Belle M., American, aged twenty years,

was admitted to the Albany Hospital suffering from pain in the head and limbs.

*Family History.*—Both parents are alive and well. Two brothers and one sister are also living and in good health. Maternal grandmother died of pulmonary tuberculosis.

*Personal History.*—Mumps and measles as a child. Began to menstruate at seventeen years; irregular. No other illness.

*Present Illness.*—Ten days ago she was taken ill with severe pain in the head, back, and limbs, accompanied by anorexia and vomiting. For the first three or four days there was some diarrhoea. Nose bleed two or three times a day for the first week.

At the time of admission she was feverish, skin hot and dry; pulse, 100, regular, and not dicrotic. The temperature in the mouth was 103° F. The patient is a well-built, well-nourished woman; face heavy and apathetic; responds to questions slowly. Pupils equal; conjunctivæ slightly diffused. Dorsum of tongue coated with thick brown fur. No glandular enlargements; no oedema. Heart and lungs normal. Abdomen somewhat distended and tympanitic. Spleen just palpable on deep inspiration; splenic dulness augmented. Liver dulness apparently increased. Some tenderness and ileocæcal gurgling on the right side. A few discrete papules similar to rose spots may be seen over the abdomen.

*March 22d.*—Condition about the same. The urine is dark amber in color, fairly clear, of acid reaction, and does not contain sugar or albumin. No casts were found in the sediment.

*25th.*—The temperature has ranged between 103° and 104° F.; the pulse between 70 and 89; the respiration between 20 and 28. Heart and lung clear; abdomen less distended.

30th.—Two days ago the temperature rose to 105° F., and has practically remained there since. Pulse 112, irregular, and dicrotic. Abdominal distention very marked; spleen easily palpable below the costal margin. Heart sounds muffled.

April 2d.—Pulse more rapid (140); temperature still up to 105° F. last evening. The patient was delirious and restless during the night. Tympanites about the same.

5th.—Delirium persists. Takes nourishment badly. Sponging has brought the temperature down some; has been passing urine frequently.

8th.—A culture taken from the median basilic vein on the 4th contains an abundant and pure growth of typhoid bacilli. The Widal reaction tried on the same day was positive; dilution, 1 to 30, in fifteen minutes; 1 to 50, in twenty-seven minutes; and 1 to 100, in an hour and five minutes.

9th.—Tip of the nose, face, and fingers markedly cyanosed; pupils dilated; temperature, last evening, 104.2° F. Coarse and fine moist râles over the base of both lungs. Abdomen tense. Death at 9.10 A.M., April 10th.

*Autopsy* (five hours after death).—Anatomical diagnosis: Typhoid fever, with ulceration and swelling of Peyer's patches; swelling of the solitary follicles of the ileum; swollen and hemorrhagic mesenteric lymph glands; broncho-pneumonia of left lung; atelectasis of right lower lobe; œdema of lungs; cloudy swelling of the liver, with focal necrosis and fatty degeneration; acute spleen tumor; acute cystitis; cystic ovary; acute laryngitis; and hemorrhagic endometritis.

Body a hundred and fifty-five centimetres long, moderately well built, and well nourished. Rigor mortis well marked in upper and lower extremities. Body still warm. The abdomen is distended, tense, and tympan-



itic on percussion. Pupils midwide and equal. Mucous membrane pale. Post-mortem lividity of the dependent parts. No subcutaneous œdema; surface of the body generally pale. Subcutaneous fat moderate in amount; muscles of a homogeneous reddish-brown color.

Brain and cord not examined. Abdomen: Peritoneal cavity free from any excess of fluid; both layers of the peritoneum are smooth, glossy, and free from any injection. Omental glands are not enlarged. The intestines are distended with gas. Foramen of Winslow patent. Liver visible for about three centimetres below the costal border. Thorax: Both pleural cavities are free from fluid. Pericardial cavity contains about one hundred cubic centimetres of clear yellow serum; both layers of the pericardium are normal in appearance. Both sides of the heart contain fluid blood, and red and chicken-fat post-mortem clots; no valvular lesions; coronary arteries smooth; myocardium normal in consistence and, on section, of a homogeneous brownish-red color. Both lungs are free from adhesions; consistence augmented. The left lung contains several broncho-pneumonic areas surrounded by œdema; the right upper and middle lobes are œdematous, the lower atelectatic. Mucous membrane of bronchi intensely congested and covered with a small quantity of muco-pus. The spleen is free from adhesions, much swollen, measuring  $16 \times 10 \times 6$  centimetres. Capsule is smooth, consistence very soft; on section, the color of the organ is dark reddish-brown; pulp is increased; trabeculæ are apparently normal in amount, and the Malpighian bodies prominent. Liver: Swollen; measures  $31 \times 22 \times 11$  centimetres. Capsule smooth, consistence normal. On section, the surface is a dull gray, with irregular areas of yellow. Here and there small pin-head-sized discrete areas of yellow are made out, presumably points of necrosis. The gall bladder is

distended with clear yellow bile; mucous membrane normal in appearance. Pancreas, adrenal glands, tonsils, ureters, tongue, and bone marrow show nothing particularly noteworthy. Kidneys: Fatty capsule moderate in amount; fibrous capsule strips off easily; surface smooth; stellate veins prominent; consistence firm. On section, the color is red; cortex markings distinct; cortex about normal in amount; glomeruli visible and congested; medulla and pelvis appear normal. The mesenteric glands are very much swollen and hemorrhagic; now and again a small area of necrosis is detected. Œsophagus: Mucous membrane injected. Stomach: Contains a small quantity of partly digested food; the walls of the viscus are not increased in thickness; mucous membrane is pale and covered with sticky mucus. Submucous pin-point to pin-head-sized hemorrhages are apparent over the surface. Pylorus normal. Intestine: Mucous membrane of the duodenum is bile-stained and congested; that of the jejunum similar in appearance; the congestion, however, is more intense in the lower part of the organ. The solitary follicles are swollen in the ileum, markedly so in its lower half; mucous membrane is congested and the serosa appears normal. The last eighteen inches of the ileum contain a few considerably swollen Peyer's patches, irregularly raised about the surface, capped by irregularly round or oval ulcers with sloping, often undermined, margins and clean bases. Appendix shows also some congestion of the mucosa, as well as a few submucous hemorrhages. Large bowel is congested in places, but otherwise is normal in appearance. Bladder: Not augmented in size; walls not increased in thickness; mucous membrane acutely congested. Tubes and ovaries adherent to one another by firm fibrous adhesions; the ovaries contain several marble-sized cysts filled with clear yellow fluid. The uterus is not enlarged;



measures  $6.5 \times 4.5 \times 3$  centimetres; peritoneal covering is smooth; walls not increased in thickness; lumen patent; endometrium smooth, and injected in the fundus of the organ. A number of pin-head-sized hemorrhages are seen in the mucosa.

*Microscopical Examination.*—The tissues were hardened in ninety-five per cent. alcohol, and the sections studied were stained with hæmatoxylin and eosin, Van Gieson's picro-acid-fuchsine, Weigert's fibrin stain, and by Flexner's methylene-blue method.

The microscopical study of sections of the myocardium shows nothing noteworthy beyond a moderate fragmentation of the muscle fibres. Sections from the lung, beyond confirmation of the macroscopical findings, add little information. The spleen presented the ordinary appearance of acute spleen tumor—hyperplasia of the lymphoid elements with intense congestion of the organ. Scattered throughout the sections are almost circular areas of necrosis. Collections of bacteria may be seen, sometimes bearing a definite relation to these areas.

Liver: The capsule is everywhere normal. The connective tissue is not increased in amount. The liver cells are greatly swollen and finely granular in appearance. At various points in the sections are more or less circular areas presenting different appearances, according to the stage of development. Some show purely a necrosis of the liver cells; others, in addition to the necrotic process, are infiltrated with round cells of the lymphoid type, while in still others the infiltration is made up of lymphoid cells and cells of an epithelioid character. No giant cells can be made out. No anatomical relation apparently exists between these areas and the portal spaces. Masses of bacilli are made out in the sections. The histological study of the mesenteric



glands, kidneys, and intestine adds little to the anatomical diagnosis, but study of the uterus shows a certain amount of desquamation of the epithelial cells of the mucosa, a moderate polynuclear and round-cell infiltration of it and the submucosa, dilatation of the small vessels, but no abnormality in the musculature of the organ.

In sections of the liver, spleen, and uterus treated by Flexner's method for staining typhoid bacilli, microorganisms morphologically similar to Eberth's bacillus were observed; in the uterus, the bacilli apparently were limited to the mucosa.

*Bacteriological Examination.*—Agar plates were made in the usual manner from the blood of the heart, bronchopneumonic areas, liver, spleen, gall bladder, mesenteric glands, bone marrow, uterine cavity, kidney and urine in the bladder at the time of the autopsy.

The plate from the uterus contained thirty-six pin-head or slightly larger, discrete, white colonies which, under the lower power, were generally round, yellowish, and finely granular. Cover slips from the growth contained short, rather thick bacilli. On media the behavior was as follows:

Agar slant: Thin, translucent, gray, filmy, moist growth on the surface of the agar.

Blood serum: Growth equally rapid to that on agar slant, and having the same general appearance.

Potato: Moist, glistening, almost invisible growth along the line of inoculation.

Gelatin stab: White growth along the line of inoculation. No liquefaction of the gelatin at the end of three weeks.

Litmus milk: No apparent change in the medium.

Bouillon and Dunham's solution became diffusely cloudy at the end of twenty-four hours. No indol reaction in Dunham's peptone solution.

Lactose and glucose agar: Stab inoculations give a somewhat scanty white growth along the line, but no evidences of gas formation.

Preparations of hanging-drop specimens from young bouillon cultures (twenty to twenty-four hours) show actively motile bacilli. In cover-glass specimens stained by Pitfield's method bacilli may be seen having five to ten flagella (peritrichal arrangement). This organism was tested with several specimens of blood from known cases of typhoid, and a positive Widal reaction was obtained in each instance in dilutions varying from 1 to 30 to 1 to 75.

Bacteriological diagnosis: *Bacillus typhosus*.

The typhoid bacillus was likewise cultivated in pure culture from the heart's blood, liver, spleen, gall bladder, mesenteric glands, bone marrow, kidney, and urine. It was also isolated from the broncho-pneumonic areas in the lung associated with a coccus identified by all the usual tests as the *Streptococcus pyogenes*.

Unlike the already published cases of this type of infection, these, as previously referred to, occurred in non-puerperal women—one in the course of atypical typhoid fever, the other as an uncommon focus in a remarkable class of typhoid cases, whose chief interest becomes centred in the absence of any anatomical lesions of the intestine. They are, however, comparable in that the source of infection lacks obvious demonstrable evidence. It is not improbable that the source for both was from within, an assumption which receives some support from the pathological findings and the bacteriological study. Over against this the presence of typhoid bacilli in the urine of the second case is important in a determination of the mode of infection; at most, in the absence of exact knowledge, attempts at explanation can be but tentative. Knowing the occur-

rence of bacillary transmission in experimental investigations, and, indeed, in human beings, it seems likely that most examples of typhoidal infection of the uterus will be included in that category of pregnant or puerperal women already suffering from typhoid fever.

### *Discussion.*

Dr. W. P. NORTHROP asked if the method of differentiating between the typhoid bacillus and the colon bacillus was sufficiently exact as to enable one to make a positive differential diagnosis.

Dr. LARTIGAU replied that the cultural features of the two organisms were ordinarily sufficiently characteristic to enable accurate differentiation. Comparative study of the behavior of the typhoid and colon bacilli to the serum of typhoid fever cases was particularly important in definitely establishing the identity of these micro-organisms.

### THE DEATH OF THE NEURON.

Dr. IRA VAN GIESON presented a preliminary communication under this title. He said that perhaps no other cell in the body so readily responded to trifling stimuli as the neuron. The crude pabulum of this cell was built up into the unstable and explosive material which made it possible for the nerve cell to do its work. There was excellent evidence that every part of the long neuraxon derived its nourishment from the nerve cell itself. Whatever material surrounded the slender filament, it was capable of storing a considerable supply of nervous energy. When the catabolic process gained the ascendancy the tip of the neuraxon no longer received its proper nourishment, and the death of the neuraxon proceeded very gradually from the tip toward the centre. When this process was



confined to the tip of the axon, its vitality might be alternately restored and destroyed. Even when the axon had been destroyed up to the cell body the latter was still able to generate energy. When this process was going on the nerve cell excreted what he had termed "metaplastm particles." This hypothesis, the speaker thought, thoroughly explained the whole process of fibre death in the nervous system. To summarize: (1) In all chronic fibre death the necrosis proceeded from the distal to the central portion; (2) the rate or intensity of death in the neuraxon was directly proportionate to the preponderance of catabolic over the anabolic process; (3) the presence and excretion of metaplastm particles in the cell body were indications of the death or impending death of the peripheral ends of the nerve fibres.

#### *Discussion.*

Dr. P. A. LEVENE said that the theory of histonus was first advanced by Max Verworn. According to this theory the anabolic and catabolic always coexisted in cells or tissues, and the condition of the tissues depended on the relation between the two processes. There were conditions when the process of anabolism was prevailing (growth), or conditions when catabolism was stronger (degeneration), and also when the two processes were in a state of equilibrium. The theory of Max Verworn was corroborated by the speaker in his researches on the developing egg. Although these facts were well known as applied to cells in general, he did not know that an application of it to cells of the nervous system had been made previously. He thought that there was not sufficient proof to consider the "metaplastm granules" anabolic elements of the cell; they could just as well be regarded as catabolic products. The latter view would be in accord with the speaker's studies regarding mucin.

Dr. VAN GIESON replied that there were several kinds of metaplasma granules, so far as they could be identified morphologically. He had identified three of these. He had at first been inclined to think that all of the metaplasma granules were anabolic, but he now took the view that they might be both of the ascending and descending kind. The theory just propounded had thus far received most extensive confirmation. Around each metaplasma particle was found a zone of clear material, and this he took to be indicative of the fact that the protoplasm was endeavoring to isolate them. The straw-colored metaplasma granules existed in the living cell, and were not precipitated bodies or artifacts. The pigment bodies he looked upon as metaplasma particles which, for some reason, had been retained within the cell for a long time and had been transformed in their chemical properties.

---

*Stated Meeting, March 14, 1900.*

EUGENE HODENPYL, M.D., PRESIDENT.

THROMBOSIS OF THE PULMONARY ARTERY; SUDDEN DEATH.

Dr. J. H. LARKIN presented specimens from a man thirty-five years of age, who had given a history of syphilis and of repeated attacks of gonorrhœa. The man had entered St. Francis' Hospital about two weeks before complaining of difficult and painful urination. Examination showed a stricture at a depth of four inches, and another in the membranous urethra. There was also some enlargement of the prostate. The next day perineal section had been done. The patient had done well for four days, and then on attempting to irrigate the bladder with hot saline solution the man suddenly became cyanotic, complained of intense pain in the chest and of dyspnoea, and in a



moment was dead. At the autopsy, having in mind the clinical diagnosis, the lungs and heart were exposed *in situ*. On opening the pulmonary artery a thrombus of large size had been found. The portion extending into the right ventricle was rounded, showing that this part had slipped down into the ventricle after having formed higher up.

FAT EMBOLISM OF THE PULMONARY ARTERY; SUDDEN DEATH.

Dr. LARKIN exhibited specimens and photographs of this case. The patient, a man aged thirty-six years, had fallen from his bicycle and injured the right hip. Examination had shown fracture of the greater trochanter of the femur and impaction of the neck. For the first few days temporary wooden splints had been used, and then a plaster spica bandage had been applied. A day or two after the man had suddenly experienced severe pain in the chest, and this had been associated with nausea and feeble heart action. He had recovered from this attack of syncope, and had done well until about one week later, when there had been another attack similar to the first one, except that it had terminated fatally. The autopsy had been performed by Dr. Norris and himself. The viscera had appeared normal. Having in mind fat embolism, portions of the viscera were taken and stained with osmic acid. In all the branches of the pulmonary artery, both the smaller capillaries and the medium-sized vessels, were found accumulations of fat globules and thrombi. It was noted at the time that the lungs were not as œdematous as had been observed in other cases of fat embolism. The microscopical appearances seemed to leave little doubt regarding the correctness of the diagnosis of fat embolism. The fractured femur was also exhibited. At the time of the autopsy the bone had been found riddled with hemorrhages.



*Discussion.*

Dr. NORRIS said that fat embolism of the pulmonary artery was very common after fractures. One observer had found, in a large series of autopsies, that there had been some fat embolism in ten per cent. of the cases. The first attack had been quite mild in the case reported, and the man had soon recovered from it. The fact that this had come on twenty-four hours after changing the dressing made it probable that the moving of the parts had been the immediate cause of the embolism. It was difficult to explain the second attack. Respiration ceased two or three minutes before the heart stopped beating, which would point rather to an affection of the blood-vessels supplying the respiratory centre.

CYSTIC DEGENERATION OF THE KIDNEYS WITH CYSTS OF  
THE LIVER.

Dr. L. A. CONNER presented specimens from a woman who had died at the New York Hospital last December. She was admitted on December 26, 1899. She was forty-four years of age. Her family and earlier personal history was unimportant. She had felt "tired" for several months. For the past six weeks she had been very weak. There was some dyspnœa on exertion. The urine had a bad odor. There were no œdema, no lumbar pain, and no hæmaturia. On admission, her temperature was 97.8° F.; respiration 32; pulse, 106. Physical examination revealed a tumor mass in the region of the left kidney and little else besides feeble heart action. The urine had a specific gravity of 1.011; it was acid, turbid, and contained 1 gm. of albumin to the litre, much pus, few red cells, and no tubercle bacilli. The patient grew steadily weaker, became stuporous, and died three days later.

At the autopsy, the heart showed moderate hypertrophy of the left ventricle—its weight was 320 gm. The heart muscle was rather soft and light colored. The valves were competent. The coronary arteries showed a somewhat anomalous distribution. The left kidney measured 19 x 8 x 6 cm., and weighed 686 gm.; the right kidney measured 15.5 x 9.5 x 7.5 cm., and weighed 472 gm. The kidneys preserved in general their usual shape, but owing to the many projecting cysts their surfaces presented somewhat the appearance of a bunch of grapes. On section both organs were seen to be composed chiefly of a multitude of cysts, which varied in diameter from 1 mm. to 2.5 cm. They were most abundant in the peripheral parts. The usual topography of the kidneys was entirely lost, and in only a few places could anything resembling kidney tissue be recognized. Several of the large cyst cavities in each kidney contained thick grumous pus. In the remaining cysts material of two fairly distinct types was seen: (1) A number of large cysts contained thick, brown, gelatinous material, which became very firm by preservation in formalin; (2) most of the smaller cysts and some of the large ones contained a thin, whitish, transparent, gelatinous fluid, which seemed to be little changed by the formalin. The pelves of the kidneys were small, and seemed compressed by the cystic kidney tissue. The ureters and renal vessels appeared normal. The bladder contained purulent urine, with mucous membrane. The liver was somewhat irregular in outline. The right lobe was prolonged downward, and was somewhat constricted at one point. It weighed 1770 gm. Scattered over the surface of the liver everywhere and projecting slightly above the surface were seen many small cysts from 2 to 10 mm. in diameter. On section of the organ these cysts were seen scattered throughout, being separated by

normal-looking liver tissue. The cysts as compared to the liver tissue made up a comparatively small part of the volume of the organ. A part of the anterior margin of the right lobe was lighter in color, tougher, and evidently contained much more fibrous tissue than did the rest of the liver. In this part the cysts were especially numerous. The left ovary was 5 cm. in diameter, and consisted chiefly of a number of good-sized clear cysts. Microscopic examination of the kidneys showed the cysts to be lined by a single layer of cuboidal epithelial cells, and to bear a very close relation to the uriniferous tubules and sometimes to the Malpighian bodies. Between the cysts in many places areas of much-damaged renal tissue could be seen. In the liver, in the same way, the cysts could be seen in process of development from the small gall ducts.

Dr. CONNER said that Richie,<sup>1</sup> of Edinburgh, after a thorough study of the subject, had drawn the following very reasonable conclusions: (1) That in cystic kidney there was an irritative lesion leading to proliferation of epithelium and also to connective-tissue changes. (There was evidence of it in simple cysts, in those which occurred in contracting kidneys, but to a much greater degree in the large polycystic kidney); (2) that in the large polycystic kidney, as in cystic disease of the mamma, the disease arose in consequence of irritation propagated through the nervous system; (3) that this disease of the kidney had no direct relation to congenital cystic kidney, which was due to an error in development; (4) that the cysts were formed from the pre-existing tubules and Malpighian bodies of the kidney, and that they were not a new formation arising out of persistent embryonic rudiments; (5) that this cystic disease was related to and

<sup>1</sup> *Reports of Laboratory of the Royal College of Physicians, Edinburgh*, vol. iv., 1894.



should be studied along with the adenomata; (6) that cystic kidney was similar in character and in origin to cystic liver. Continuing, the speaker said that a number of other writers had concluded that this condition had nothing to do with congenital cystic kidney, and that it should be classed along with the adenomata. It had been suggested that they should be called multilocular adeno-cystomata. Richie had tabulated eighty-eight cases, in twenty-one of which it had been associated with cystic liver, in two with cystic ovary, in one with cystic uterus, and in one with cystic thyroid. In all but two cases both kidneys had been involved. Hypertrophy of the heart had been noted in twenty-nine out of thirty-nine cases. The average age was forty-five years, the youngest patient being twenty-three and the oldest eighty-eight years. As to sex, there were slightly more males than females. In three cases the symptoms had existed for over fifteen years; in seventeen for over one year. Thirty out of seventy-eight had given some renal symptoms, such as pain, hæmaturia, and oedema. Of the seventy-two cases, thirty-eight patients died with symptoms of uræmic coma, and eight of cerebral hemorrhage. The microscope had shown in the kidney islands of greatly damaged renal tissue between the cysts, and the cysts themselves bore close relation to the uriniferous tubules and sometimes to the Malpighian bodies. In the liver, the close relation to the small gall ducts was evident.

#### *Discussion.*

Dr. E. HODENPYL said he was very much astonished at the citations made by the last speaker, for it seemed to him that it had been quite generally accepted that such kidneys as these were of congenital origin. He had seen the condition a number of times in the newly born, and the speci-

men just presented reminded him forcibly of a similar one that he had shown to this Society a year or two before. He saw no reason for calling these cysts tumors. From the fact that children were born with these it seemed reasonable to look upon them as congenital.

Dr. JAMES EWING said he had been surprised at the statements quoted from Richie, and he would like to know more regarding the grounds upon which these cysts were declared to be tumors. He would like to know what was the character of the lining of these cysts. The kidneys just presented seemed to exhibit much more of the renal tissue than in the cases he had seen in the newly born.

Dr. CONNER replied that cases of congenital cystic kidney resulted fatally after a comparatively short time, and, so far as he knew, the condition had not been traced from early infancy into adult life. Richie and others had found many evidences of tumor formation, and had given their reasoning in detail. The cysts in this case had been lined with cuboidal epithelium.

#### A CASE OF PERFORATED ULCER OF THE DUODENUM.

Dr. A. J. LARTIGAU presented a specimen of perforated duodenal ulcer. The specimen was from a man, sixty-four years of age, who had entered the Roosevelt Hospital on January 17th complaining of pain in the right iliac fossa. One week previously he had been taken ill with pain in the region of the umbilicus. On admission, the diagnosis had been made of general peritonitis, probably from perforation of the appendix. On opening the peritoneal cavity a large quantity of thin fetid pus had escaped and the intestine and appendix in that region had been found normal. Further search had revealed the presence of a perforation of the bowel, and of adhesions to the liver. The operation had not been pursued further, and the man



had died eight days afterward. The autopsy had been made five hours after death, and the anatomical diagnosis had been purulent peritonitis, chronic diffuse nephritis, and miliary tuberculosis of the lungs, spleen and ileum. The duodenum was adherent to the under surface of the right lobe of the liver. The duodenal ulcer was unusually large, measuring 3 x 2 cm. As a rule, the pancreas was the protecting barrier, but in this instance the liver had formed the protecting wall. The microscopical appearance of the ulcer was similar to that of the ordinary round ulcer of the stomach.

Dr. LARTIGAU remarked that while these ulcers were usually circular, the one he had presented was oval. Most of the duodenal ulcers closely hugged the pylorus, as in both these specimens.

Dr. J. H. LARKIN presented a specimen of perforated duodenal ulcer taken from a man twenty-eight years old, who had been ill three days before coming to hospital. His condition at that time had been very bad, but an operation had been immediately undertaken. An extensive peritonitis had been found, and the peritoneal cavity contained much fetid pus. The appendix was normal. The perforation had been found at the site of a small ulcer in the posterior wall of the duodenum.

#### MICROSCOPIC DEMONSTRATION OF "VACCINE BODIES."

Dr. A. W. WILLIAMS gave this demonstration. She said that the principal point of interest in regard to the so-called "vaccine bodies" was that their nature had not yet been determined. Most observers, among them L. and E. Pfeiffer, Guarnieri von Wasielewski, Von Sicherer and Kourloff, considered them micro-organisms, placing them among the rhizopoda in the group Sarcodina of the protozoa, and basing their belief upon the facts that in the



fresh tissue they showed amœboid movements, that some possessed a more refractive central spot (nucleus) and showed evidences of division by fission, that they had been found in no other disease, and that they could be accounted for in no other way. On the other hand, Ferroni and Massari, Salmon, Huckel, and others said that these appearances were due to products of a degeneration, either intra-cellular, proceeding, as some said, from the nucleus of the epithelial cells, others from the epithelial cell body—the centrosome perhaps; or extra-cellular, from the leucocytes, the epithelial cells then taking up the degenerated particles. Such degeneration, they stated, was probably specific, and might be produced by an unknown micro-organism. The specimens under the microscope were a section from a rabbit's cornea, hardened forty-eight hours after inoculation with vaccine virus, and one from an isolated vaccine vesicle on the skin of a calf six days after inoculation. The section from the cornea was hardened in bichloride of mercury and stained by Heidenhain's method. The portion under the microscope showed the edge of the area of inoculation. Immediately about the point of inoculation the epithelial cells had fallen off, leaving only the lower layers of epithelium. The vaccine bodies lying, one or two generally, sometimes more, in the body of the epithelial cell, were stained a more or less homogeneous brown-black; the bodies of the epithelial cells were a light yellow, and their nuclei were an irregular faint gray. The specimen of calf-skin was hardened in absolute alcohol and stained with hæmatoxylin (Delafield's) and eosin. In the field under the microscope was a sebaceous gland in which the bodies showed more plainly on account of the large size of the epithelial cells. The vaccine bodies were here stained a more or less homogeneous purple, the nuclei of the epithelial cells a darker irregular purple, and their bodies a light pink. The nuclei

of the leucocytes which were present in moderate numbers about the gland, and occasionally within it between the epithelial cells, were stained also a dark purple.

#### A CASE OF TRICHINOSIS.

Dr. HARLOW BROOKS reported this case from the fourth medical division of Bellevue Hospital, in the service of Dr. Lambert. After eating sausage a man had begun to complain of malaise and muscular soreness. Microscopical examination of several sections from his muscles had showed a myositis, but no trichinæ. Subsequently a large piece of muscle had been excised under cocaine anæsthesia from the junction of the belly of the biceps muscle with its tendon. From this specimen one trichina had been found about to become encapsulated. The fæces had been carefully examined, but no trichinæ were found. There had been 18,000 leucocytes and ten per cent. of eosinophiles at the time of his admission to hospital on February 2d. On February 12th he had had forty-four per cent. of eosinophiles and a proportionate leucocytosis. The percentage of eosinophiles had steadily increased up to a maximum of eighty-three per cent., and then had slowly declined. At the present time the percentage of eosinophiles was fifteen per cent. It had seemed to him that these eosinophiles differed somewhat from the cells ordinarily called by this name. They were possibly a transition form.

#### *Discussion.*

Dr. LARTIGAU said that he had recently had occasion to examine specimens of blood from various cases of a small epidemic of trichinosis investigated by Dr. George Blumer, of Albany, N. Y. The percentage of eosinophiles in none of these cases had been so high as that just reported. As recovery had taken place the eosinophilia had



declined; at the end of two months Dr. Blumer found these cells still increased in number, although much diminished. In many, but not all, cases, a decrease in the small mononuclears was observed. It was worthy of note that some of the severest cases did not necessarily correspond with the degree of eosinophilia, some of the mild cases showing a greater increase than the clinically more severe ones.

Dr. EWING said that the case reported by Dr. Brooks gave the highest percentage of eosinophiles on record. In this connection he would refer to the possible aid to diagnosis afforded by the increase in the percentage of eosinophiles. A marked increase in these cells might furnish grounds for suspecting trichinosis, but nothing more than this except where it was associated with an exudative myositis. When this combination was present, one was justified in making a diagnosis of trichinosis. He had carefully examined the eosinophile granules in this case, and had found that many of them were smaller than the usual type, but reacted the same with staining agents. He had been unable to find any evidence that they were transition forms between eosinophiles and neutrophile granules. If there was a transition here, it was probably between the eosinophile and basophile granules. The case was exceedingly interesting because of the influence which a parasite like the trichina seemed to have upon the eosinophile cells.

Dr. LARTIGAU remarked that notwithstanding the fact that the eosinophilia might be attributed to a number of different causes, he regarded a marked increase in the eosinophiles as extremely suggestive of trichinosis. He knew of several unpublished sporadic cases in which the diagnosis had first been made wholly by the differential blood count, and subsequently verified by finding the trichinæ.



A CASE OF CHYLOUS URINE AND FILARIA SANGUINIS  
HOMINIS.

Dr. M. NICOLL, Jr., reported this case, and presented the patient, a young man, aged nineteen years, a native of Santa Cruz. Six months ago, without previous symptoms, he had begun to pass creamy urine. After passing this daily for two months, he had noted that the urine was pinkish, and then that there was a momentary retention, relieved by the passage of a small clot of blood. For the past four months he had had in the left groin varicose glands, which gave rise to an appearance resembling a hernia. The urine was now pink and creamy, and contained fat cells. He had been unable to detect the filaria in the urine, though they were probably present. Dr. Nicoll also exhibited under the microscope the embryonal filaria—the variety found in the blood.

## A CASE OF SOLITARY TUBERCLE OF THE HEART.

Dr. E. DUNHAM reported this case, which occurred in a person aged twenty-one years, in the service of Dr. John W. Brannan, at Bellevue Hospital. There was a solitary tubercle about the size of a pea on the inner surface of the wall of the left auricle, just beneath the endocardium. There was a miliary tuberculosis in the lungs, several large tubercles in the brain, and a solitary tubercle in one of the kidneys. It was rather difficult to explain the occurrence of the solitary tubercle immediately underneath the endocardium in the auricle. A number of sections had been made and examined, and in every one it had been possible to demonstrate from one to six tubercle bacilli, all within giant cells.

*Discussion.*

Dr. HODENPYL remarked that there were a number of similar cases of solitary tubercle of the heart on record.

A CASE OF ABERRANT SUPRARENAL BODY WITH PACHYMEN-  
INGITIS HEMORRHAGICA INTERNA.

Dr. F. C. WOOD reported this case; also a case of aneurism of the aorta with unusual lesions of the lung, and a case of tuberculosis with multiple sarcomatosis.

Some notes were then read by Dr. GEORGE C. FREEBORN on

THE PREPARATION OF HÆMATEIN STAINING SOLUTIONS  
AND ON THE TECHNIQUE OF STAINING.

Up to a few years ago all formulæ for the preparation of hæmatoxylin solutions, especially those containing alum, ended with the following directions: "Allow to stand in the light from eight to ten days before using." This was for the purpose of allowing the staining fluid to "ripen," as it is well known that freshly prepared solutions are of no use for staining.

This "ripening process" was found to be due to the oxidation of the contained hæmatoxylin and the formation of hæmatein, through the absorption of oxygen from the air. Paul Mayer and Unna found that this oxidation could be brought about immediately by the addition of a small quantity of hydrogen peroxide; Hansen, by the addition of potassium permanganate. We have hastened the process by passing air through the solution, using air compressed by the usual spray apparatus.

All hæmatoxylin solutions deteriorate in the course of time, the oxidation process continuing until precipitates form and the solution becomes colorless. Unna found that this over "ripening" could be delayed by the addition of a small quantity of sulphur. Paul Mayer used glycerin, and Gage, believing this to be due to the action of bacteria, sterilized his alum solution and then added chloral hydrate as a preservative. Owing to the inves-

tigations of Paul Mayer we are now in a position whereby hæmatein staining solutions can be quickly made, without the long "ripening" process.

Hæmatein, which Mayer uses in place of hæmatoxylin, comes in the form of dark greenish crystals or powder, and is freely soluble in alcohol and water. The commercial form is not always pure, and owing to this, hæmateate of ammonia has been recommended in the place of hæmatein. This salt is also found in commerce, but of doubtful purity. It can be readily made in the laboratory as follows: One gramme of hæmatoxylin crystals is dissolved, by the aid of heat, in 20 c.c. of distilled water and filtered. To this solution 1 c.c. of ammonium hydrate (sp. gr. 0.875) is added, and the dark purple fluid placed in a shallow porcelain dish, of such a size that the layer of fluid shall not exceed one half a centimetre in depth. The dish is covered with filter paper, to exclude dust, and the solution allowed to evaporate at the room temperature. The hæmateate of ammonia about equals in weight the hæmatoxylin used. In the preparation of this salt no metal instruments should be used until the powder is absolutely dry.

Mayer gives formulæ for three different solutions of hæmatein as follows:

*Hæmalum*.—Dissolve 1 gm. of hæmatein or hæmateate of ammonia in 50 c.c. of 90 per cent. alcohol, and add this to 100 cc.c. of a 5 per cent. aqueous solution of potash alum, filter, and add a crystal of thymol to prevent the formation of moulds. This fluid has a color resembling that of borax carmine. It may be used for staining in full strength, or it may be diluted with weak alum solution. In full strength it stains sections in about two minutes.

*Acid Hæmalum*.—This is prepared by adding two per cent. of hydric acetate to the above hæmalum solution.



After staining, wash well in water to bring out the bluish tint of the nuclei. This solution is decidedly less diffuse in staining and keeps better than the hæmalum.

*Glycerin Hæmalum.*—The above solutions of hæmatein do not keep well. To correct this, Mayer adds glycerin as a preservative. The solution is made as follows: Rub up 0.4 gm. of hæmatein in a mortar with a small quantity of glycerin; dissolve 5 gms. of potash alum, 70 c.c. of distilled water, and add 30 c.c. of glycerin. This mixture is gradually added to the hæmatein solution. This stain keeps well, but is somewhat slow in its staining.

During the last year we have been using hæmatein in place of hæmatoxylin with the most satisfactory results. The staining fluid we have used is one made after a combination of Mayer's and Gage's methods. Our solution is made as follows: A 5 per cent. solution of potash alum is made in distilled water and boiled, or, what is better, placed in a steam sterilizer from one to one and a half hours. While this solution is warm, 1 per cent. of hæmatein, dissolved in a small quantity of alcohol, is added (it is our practice to keep a 1 per cent. alcoholic solution of hæmatein as a stock solution). After the fluid has cooled, add 2 gms. of chloral hydrate for each 100 c.c. of the staining fluid. This stain keeps well, and in full strength stains sections in from two to three minutes. It may be used diluted with distilled water, when the staining requires a longer time.

*Staining Technique.*—Soon after the introduction of the sieve dishes of Steinach, we employed them in staining. These dishes are of glass, the bottoms being perforated with a number of holes. These holes are comparatively few in number, consequently the fluids do not drain away rapidly, also being of glass they soon become broken. For these sieve dishes we have substituted the ordinary tea-strainers. These are made of fine meshed,

tinned wire; they are of various sizes and shapes, and are cheap. In the use of these strainers we proceed as follows: The sections, in alcohol, are poured into one of these strainers, the alcohol draining away quickly. The sieve part of the strainer is then immersed in water contained in a cylindrical-shaped vessel, and of such a diameter that the rim of the strainer will rest on the top. The strainer is raised and lowered several times, so that the alcohol may be thoroughly removed from the sections. The strainer is then transferred to a vessel containing the staining fluid, then successively to two containing water, then to one containing strong alcohol, and finally to one containing eosin alcohol to complete the dehydration, and the staining with eosin.

The eosin alcohol used by us is prepared by the 'method of Sharp, and the eosin added to this in the proportion of one to fifteen hundred. By means of Sharp's method the ordinary alcohol is very much increased in strength, in many cases being brought up from 99.6 per cent. to 99.8 per cent. This alcohol is prepared as follows: Cupric sulphate is pulverized and heated at a temperature of  $100^{\circ}$  C. until all the water of crystallization is driven off, and a white powder is the result. This powder, anhydrous cupric sulphate, is placed in a glass-stoppered bottle, until the bottle is about one third full, and ordinary alcohol added. After standing for twenty-four hours, the surface of the copper will have become decidedly blue, from the absorption of the water from the alcohol. Test made at this time will show the alcohol to be over 99 per cent. Fresh alcohol is added, from time to time, until all of the copper has become blue in color; now it is no longer of any use for dehydration. The supernatant alcohol is now poured off, and the bottle placed in a hot-air sterilizer at a temperature of  $100^{\circ}$  C., until the cupric sulphate is again rendered anhydrous.



The eosin is prepared by the method of Fisher. The eosins of commerce vary very much in their staining properties, so that the same results are hardly ever obtained with two different samples. By the use of Fisher's method an eosin is obtained which gives almost constant results. Make a saturated solution in water with Grübler's water-soluble eosin, and precipitate this solution with hydric chloride, adding a slight excess of the acid. Bring the precipitate on a filter, and wash with water until the filtrate begins to come away slightly tinged with the eosin. This indicates that all of the acid has been washed out. Allow the precipitate to dry; powder, and preserve for use. The eosin prepared by this method is several shades lighter in color than the eosin of commerce. It is decidedly more selective in its staining, and gives a more transparent stain. It is quite freely soluble in alcohol, but slightly in water.

Personally, we have discontinued the use of eosin alcohol in our staining technique, adding the eosin to the clearing oil. We keep a saturated alcoholic solution of the above eosin in stock, and add say 5 to 10 drops of this to the oil; as the staining of the oil becomes weak this addition of the alcoholic eosin is repeated. In our experience, this gives a clearer and more selective stain.

For clearing purposes we now use a mixture of the French white oil of thyme and oil of *Origanum creticium*. This mixture acts as well as the pure oil of organum, and is decidedly less expensive. We mix one part of oil of *Origanum creticium* with three parts of the oil of thyme, add a considerable quantity of powdered chalk, and after twenty-four hours filter. Many of the oils used for clearing have a more or less acid reaction, which in many instances will bleach out the hæmatein stain. The treatment of the oil with chalk corrects this acidity.



*Discussion.*

Dr. BROOKS asked if Dr. Freeborn found that the acid in the acid eosin injured the clearness of the hæmatoxylin stain.

Dr. FREEBORN replied that if the precipitated eosin was washed until the wash-water became tinged, one could be sure that all of the acid had been removed. He had never known it to interfere with the hæmatoxylin stain.

Dr. BROOKS asked if the sections could be left for a long time in the eosin oil without danger of over-staining.

Dr. FREEBORN replied that he had left them in so long as ten days, and had not been able to observe any detriment from so doing.

Dr. EWING said that for the last six months he had tried all the purified eosins he could find in the market, yet he had not been able to make a good blood preparation with any of them. In order to get a good selective stain for blood he was sure it was necessary to improve on the ordinary eosins now on the market.

Dr. FREEBORN said that the eosin employed by him had been procured from Grübler, and was known as "water-soluble eosin." The acid was added until a precipitate no longer formed. He had not tried the effect of using an excess of the acid.

---

*Special Meeting, March 29, 1900.*

EUGENE HODENPYL, M.D., PRESIDENT.

A CASE OF EPITHELIOMA OF THE ŒSOPHAGUS WITH STENOSIS  
OF THE TRACHEA AND SUFFOCATION.

Dr. J. H. LARKIN presented specimens removed from a man, sixty-three years of age, who had always been in excellent health up to five days before operation. At that

time he had experienced a slight dyspnoea at first, and this had increased rapidly until at the time of his admission he was deeply cyanosed. Tracheotomy had been done immediately. On opening the trachea and endeavoring to pass a tube, some difficulty had been encountered. A small tube had been passed without giving him much relief. On the following day the tube had caused so much distress that it had to be removed. The man only lived forty-eight hours after the tracheotomy. At the autopsy all of the abdominal viscera had been found normal, and the lungs also with the exception of a slight oedema. About three inches above the cardiac end of the oesophagus was a small, slightly raised area which, on microscopical examination, showed epithelioma. High up in the trachea, and about one inch below the tracheotomy wound, and about one inch and a half above the bifurcation of the bronchi, was a marked stenosis of the trachea. The cause of this was a tracheal lymph node which had enlarged and grown through the tracheal rings and had occluded the trachea at that point. Microscopical examination had shown this to be a metastatic epithelioma of the lymph node. On account of the pressure on, and erosion of, the tracheal rings a secondary suppurative process had taken place in the lymph node. Death had resulted from slow asphyxia and oedema of the lungs.

#### OSTEOCARCINOMA OF THE BREAST.

Dr. LARKIN also presented a tumor which had been removed from a woman of middle age by Dr. Kammerer. Five months ago she had first noticed a slight enlargement of the breast. At the time of the operation the growth was supposed to be carcinoma, but after its removal it was found exceedingly difficult to cut into the tumor. Further examination showed the tumor to be a well circumscribed growth, and to have a limiting wall.

At the periphery it was somewhat spongy, but toward the centre was a large and somewhat grayish elastic area which was undoubtedly cartilage. In the centre were nodules made up of true bone. Microscopical examination showed at the periphery a typical picture of carcinoma, but the carcinomatous element diminished as one proceeded toward the centre. In the latter location were bone and cartilage. A search through the literature showed that few tumors of this kind had been reported. The speaker said that cases of calcification of tumors of the breast had been reported in Virchow's *Archiv* for 1897. Osteosarcomata of the breast had also been described, but very few osteocarcinomata had been reported. Coen, of Boulogne, had reported a case almost identical with the one just described. In that case the woman was forty-five years of age, and the clinical history had presented nothing unusual. A case of osteo-chondro-carcinoma had been reported by Hacke. Lesser had also reported a similar case, as had Leon, of Paris. Astley Cooper, in his book on *Diseases of the Breast*, also reported such a case, but there was some doubt about the correctness of the diagnosis. Dr. Larkin raised the question as to whether this tumor had been at first an osteochondroma, or a combined growth of carcinoma with a secondary metaplasia of the stroma into osteoid tissue. As in the periphery metaplastic changes could be seen in the alveoli, it seemed to him reasonable to look upon this tumor as having been first a carcinoma, and that secondarily there had been a metaplasia of the fibrous tissue, resulting first in cartilage, and ultimately in bone.

#### *Discussion.*

Dr. LARTIGAU said that pathologists had hesitated to accept the belief that true osteoid tumors of the breast



existed primarily as such. The tendency was to regard these neoformations as ossifying enchondromata. A study of the literature showed that enchondromata were very common in the mammæ of bitches. Why this should be so more than in other animals he was unable to say, nor had any attempt been made, so far as he knew, to elucidate this question.

#### A CASE OF ACTINOMYCOSIS.

Dr. F. S. MANDLEBAUM exhibited under the microscope a section from a case of actinomycosis removed from a woman of thirty-four years, a Russian, who had been admitted to hospital on January 25, 1900, with a history dating back five months, and beginning with the development of two localized points of tenderness on the back to the right of the spine. Three months after the onset of pain, small swellings without redness had appeared at these points. Three weeks later, or five weeks before admission, these had been excised, and in one week a swelling had appeared in the right axilla and above the clavicle on the same side. On admission, these tumors had been the size of a small apple, and tender and painful, but the skin had not apparently been involved. Near the right scapula was a discharging sinus, and two inches below this another sinus. The entire supraclavicular triangle was occupied by a mass, in places fluctuating. Physical examination of the chest showed anteriorly dullness over the first right interspace, with exaggerated breathing, and posteriorly marked dullness and tenderness on percussion from the apex downward to the spine of the scapula. Over this vocal fremitus was increased. The woman was emaciated and six months pregnant. One week after admission abscesses in the neck and axilla had been incised, and a large quantity of pus, streaked with blood and containing numerous small, yellowish par-

ticles, had been evacuated. She had received iodide of potassium internally, and had improved considerably while in the hospital. Dr. Mandlebaum said that he had taken small drops of the pus and fixed them in five per cent. formalin solution. The small pieces were then hardened in alcohol, embedded in celloidin, and sections made. The microscope showed these small particles to be actinomyces. He had stained them with many different stains, and had found that the best results were obtained with Plant's stain. This consists in staining the section for ten to fifteen minutes in a solution of carbol-fuchsin; then washing in water; transferring to a saturated alcoholic solution of picric acid for five minutes; decolorizing in fifty per cent. alcohol for fifteen minutes, and finally passing through absolute alcohol, oil of cloves, and into balsam. With the Gram method in only one instance had he been able to demonstrate the clubbing, although this was brought out well by following with eosin. The teeth and mouth were normal, and the primary focus was supposed to have been somewhere in the chest.

HÆMATOMA OF THE RIGHT SUPRARENAL GLAND WITH  
RUPTURE INTO THE PERITONEAL CAVITY OF A CHILD  
OF TEN DAYS, WITH A PATENT FORAMEN OVALE,  
PATULOUS DUCTUS ARTERIOSUS, AND STENOSIS ISTHMI  
AORTÆ.

Dr. CHARLES NORRIS reported this case. The child had been brought to Bellevue Hospital with no further history than that it had been blue since birth. It had died very suddenly a few minutes after entering the hospital. On autopsy, the abdominal cavity had been found filled with blood, and the intestines all matted together by fairly firm adhesions. On the superior surface of the suprarenal, represented by a more or less organized blood-clot of a considerable size, was an opening two inches



in diameter, through which the blood had escaped into the abdominal cavity. On examining the foramen ovale it had been found nearly closed. The ductus arteriosus as it entered the aorta presented a funnel-shaped opening. The speaker said that hæmatomata of the suprarenal were rarely as large as the one just presented. This was the second case of the kind that he had met with. He had been able to find only one or two instances reported in literature in which rupture had taken place into the peritoneal cavity. One observer had reported twenty-six cases in which there had been small hemorrhages into the suprarenal in a series of over one hundred autopsies on still-born infants. In two of the cases the hemorrhage had started in the cortex. The blood more commonly escaped into the perinephritic tissues. It was more often met with after pelvic than podalic presentations, and after difficult labor.

*Discussion.*

Dr. LARKIN said that having had an opportunity of examining a number of still-born children he had met with four or five such hæmatomata. His experience confirmed what had been said about their being more frequent after difficult labors, and with pelvic presentations. He had never before seen one rupture into the peritoneum, but had seen the blood escape behind the peritoneum, and had dissected out the latter down as far as the bladder.

Dr. EUGENE HODENPYL presented several specimens, as follows:

A PORTION OF THE ŒSOPHAGUS SHOWING VARICOSE DILATATION OF THE VEINS.

This specimen had been taken from a man who was suffering from cirrhosis of the liver, and who died of a



profuse gastro-intestinal hemorrhage. The œsophagus in the fresh state showed a number of very large and tortuous veins in the lower portion, at least one of which was ruptured. Preble, of Chicago, had found that in about eighty cases of fatal hemorrhage occurring in the course of cirrhosis of the liver, the bleeding arose from the varicose veins in the lower two-thirds of the œsophagus. This accident occurred in atrophic cirrhosis rather than when the liver was of normal size or enlarged. Attention was called to the fact that the venous circulation of the œsophagus was part of the systemic circulation, while that of the cardiac end of the stomach belonged to the portal system, and that there was a poor collateral circulation between the two. There might be also a negative pressure on the œsophageal veins during expiration.

#### PNEUMOTHORAX.

The next specimen had been taken from the gripman of a car, who had been suddenly seized with severe pain in the right chest while at his work. He had been hurriedly removed to a hospital, and examination had revealed the presence of air in the right pleural cavity. He had died in a few hours. In spite of the man's excellent physique the autopsy showed quite advanced tuberculosis of both lungs, and the kidneys were studded with miliary tubercles. There was also a moderate tuberculous enteritis. The liver was found forced almost entirely below the free border of the ribs, and the diaphragm bulged downward. On puncture of the diaphragm below a water level, a considerable quantity of air escaped. The right lung was forced into the bottom of the pleural cavity against the vertebral column. This lung was entirely free from adhesions, although the pleura over the posterior portion of the apex was markedly thickened.

The opening was so minute as to be discoverable only after forcing air through the bronchi.

#### A TUBERCULOUS APPENDIX.

The third specimen had been taken from a man who had died of pulmonary tuberculosis with quite marked tuberculosis of the intestine. The latter was very markedly dilated, and it was so dark as to suggest gangrene. There were two constrictions in it. The miliary tubercles could be distinctly seen through the serosa. In tuberculosis of the appendix, as well as in typhoid ulceration of the appendix, the speaker said, it was worthy of note that the usual symptoms of appendicitis were not present.

#### PRIMARY CARCINOMA OF THE PANCREAS WITH SECONDARY CARCINOMATOUS NODULES IN THE LIVER.

The specimens from this case had been taken from a person who had suffered but slightly, and had been ill only a few weeks. He was an alcoholic subject who had presented but few symptoms, but had died shortly after entering the hospital. At the autopsy immense deposits of fat were found throughout the body. Occupying the head of the pancreas was a large carcinomatous mass. The liver was studded with carcinomatous nodules. Examination of the heart showed dilatation of the right side of this organ and incompetence of the tricuspid valve.

#### TWO CASES OF MULTIPLE SPURIOUS DIVERTICULA OF THE INTESTINE.

The specimens from two cases of this kind were exhibited. In the first, the duodenum and upper portion of the jejunum was the seat of a number of thin-walled



cysts varying in size from that of a pea to that of an egg. The second specimen was from a case of papilloma of the peritoneal cavity. There were strictured areas in various parts of the colon, and seventy-nine diverticula were found in the intestine, principally in the lower end of the colon. Roth, in 1872, had described five cases of diverticula of the duodenum, in all of which the diverticula had been small and not over three in number. In 1894, an article on this subject had been published in Virchow's *Archiv* by Edel. Here the cases had been divided into two classes—congenital and acquired. Meckel's diverticulum alone constituted the first class. All three coats were present in the congenital form, whereas the muscular coat was missing in the acquired variety. The most common situation for these false diverticula was in the colon; next in order of frequency, and usually along the attached border, came the duodenum. The number of these diverticula varied from one up to two hundred or more. As a rule, they were small. The cause of the condition seemed to be a separation of the fibres of the muscular coat, so that, in one sense, they might be regarded as herniæ of the mucous membrane into the serosa. The pressure of scybalous masses, the presence of gall-stones, circumscribed or diffuse peritonitis, constipation, and carcinoma were all causes. It had been noted that these diverticula were almost always found empty at autopsy.

The paper of the evening was read by Dr. AUGUST JEROME LARTIGAU on

#### HYPERPLASTIC TUBERCULOSIS OF THE INTESTINE.

During the last ten or twelve years a few writers, particularly French and German observers, have from time to time called attention to the existence of a form of



tuberculosis affecting various segments of the intestinal tube, and characterized by a variable but usually a considerable degree of thickening of the wall of the intestine. So definite and constant is this distinguishing characteristic that the type has been habitually referred to by French writers as a *tuberculose intestinale à forme hypertrophique*. Ordinarily localized in the ileo-cæcal region, but likewise affecting other regions more infrequently, the process affects a disease of long duration, the most conspicuous feature being the more or less extensive formation of fibrous and tuberculous granulation tissue in the involved parts. This is often of such a character that the inflammatory hyperplasia or "pseudo-neoplasm" (Benoit) may easily be mistaken clinically for tumor-formation of the locality, resembling carcinoma. A condition more or less similar has been described in the larynx; its clinical presentation is also that of cancer of the parts. In serous membranes hyperplastic tuberculosis is also observed; the peritoneum, pleura, and especially the joints are affected in this way. Recent studies further show a greater or less parallel process in a group of cases diagnosticated Hodgkin's disease, but which fuller investigations have shown to be incited by the bacillus tuberculosis. In processes of this kind the tubercle bacillus and its products incite fibrous hyperplasia rather than caseation and necrosis.

Since the first detailed study of this peculiar type of intestinal tuberculosis by Hartmann and Pilliet in 1891, it has been the source of considerable interest and some discussion. To the best of my knowledge the earliest serious investigation of this anatomical type begins with these writers, the one investigating the pathological side, the other the clinical features of the disease.

The specimen of hyperplastic tuberculosis of the intestine which I was able to study is one of the highest in-

terest. The length of gut involved makes it unique in the literature of this affection; and the absence of distinct stenosis and apparent ulceration of the mucous membrane anywhere in the intestine adds further interest to the report. For the clinical story of the disease I am indebted to Dr. J. L. Schoolcraft, of Schenectady, N. Y.

Simon S., aged forty-nine years, was a business man until shortly after the onset of his trouble, when failing health necessitated inactivity. The family history shows no evidence of tuberculosis. When a child he suffered from some of the acute infectious diseases of childhood; otherwise his health had always been excellent. No history of lues, typhoid, or dysentery.

About three years before his death (1895) he is said to have lost in weight and to have become gradually weaker and weaker, without apparent cause; so much so that walking became too fatiguing. On the slightest exertion a tendency to fainting had been manifest. Co-existent with this loss of strength there was noticed a change in the color of the skin; from a rather fair skin the color passed to a dark brown, so much so that one year after the commencement of the trouble the tint had become quite bronze-like. The distribution of the pigmentation was universal, rather than patchy; perhaps most accentuated on the exposed surfaces. Rather early (two months from start) he began to suffer from cramp-like pains all over the abdomen; the patient had been unable to localize them to any particular region. At times he fancied they were most intense in the right iliac region. Generally the attacks would last for so short a period as five minutes; often, especially later (during last year of sickness), as long as one half hour or longer. For the most part the cramps came after irregular intervals of quiescence; at first, not over one a week; then later so frequently as several (four to eight) times a day. These



attacks were wholly independent of the taking of food; sometimes the onset occurred shortly thereafter, more often without any reference thereto. Vomiting occurred from time to time, being especially liable to be associated with the attacks of abdominal pain. Pressure had no effect in increasing or diminishing the severity of the pain. With time this whole chain of symptoms increased in severity. The appetite had been generally poor, the bowels irregular—alternating diarrhoea and constipation. A tendency to looseness, however, had been shown during the entire sickness. No mass had at any time been felt by the patient in the abdomen; but a sensation of vague discomfort and heaviness had been almost constantly present. Nothing abnormal had been noticed about the stools. Blood had never been noticed.

About one year and a half before his death he came under medical observation. At this time he was a poorly nourished man of slim build, complaining of great weakness and loss of flesh. The patient had lost about thirty pounds in weight in the first year of his sickness. The temperature was then normal; the skin of the face and hands, as well as that of the trunk, of a deep brownish color; the same was true of the body generally; in no wise was this patchy in character, nor was the pigmentation distinctly bronze-like. The mucous membrane of the eyes and mouth was pale and free from pigmentation. The chest was well developed, and the heart and lungs appeared normal. Examination of the abdomen revealed nothing; except on deep palpation tenderness was elicited, seemingly most marked in the right iliac fossa; the spleen was not palpable, nor was the liver enlarged. Superficial glands nowhere enlarged. Urine normal. When seen for the first time about eighteen months before death a diagnosis of Addison's disease was ventured. Arsenic, cod-liver oil, bone-marrow, and a very liberal



and nourishing diet were prescribed; and he was sent to the country. For a time some improvement took place; he appeared to eat better, increased some in weight, and the abdominal disturbances improved considerably. This continued for about three or four months. Then, instead of further amelioration the condition became more aggravated, with accentuation of all symptoms. Temperature observations during this time showed a fairly constant evening rise of one to two degrees F. About two months before death, fainting fits occurred on slightest exertion, until weakness necessitated remaining in bed for the last few weeks of life. Finally, some days before dissolution, symptoms of right-sided lobar pneumonia developed, and he died December 31, 1898.

The autopsy was performed on January 1, 1900, about twelve hours after death. The body had been injected with embalming fluid containing formalin, hence no cultures were made from the case. The notes from the autopsy-protocol are as follows:

*Anatomical Diagnosis* (revised): Hyperplastic tuberculosis of the small and large intestine; lobar pneumonia (gray hepatization) of right lower lobe; oedema of lungs; tuberculosis of suprarenal capsules; tuberculosis of mesenteric lymph nodes; acute splenic tumor; cloudy swelling of the liver; slight chronic diffuse nephritis.

Body 160 cm. long, sparely built; moderately emaciated man. Rigor mortis absent in both extremities. No subcutaneous oedema of lower extremities. Surface of body pale and universally of a dark brownish color, more particularly pronounced on face and other exposed cutaneous surfaces. Nowhere is this patchy in character, nor does it at any point suggest bronzing. The appearance is more distinctly that produced by exposure to the sun. Mucous membrane of mouth free from abnormal pigmentation. Pupils moderately dilated and equal. Mucous mem-

branes pale. Post-mortem lividity dependent parts. Subcutaneous fat small in amount. Muscles of thorax and abdomen poorly developed and pale.

*Peritoneal Cavity*.—Both layers smooth and normal. *Foramen of Winslow* patent. Congenital absence of the appendix. *Omentum* delicate. *Omental glands* not enlarged. *Diaphragm*, right side fifth rib. Left side fifth space. Right lobe of liver reaches 5 cm. below costal margin; stomach not visible. Both pleural cavities free from fluid.

*Pericardium*.—Both layers smooth. Cavity contains a small quantity of clear yellow fluid.

*Heart*.—Small. Distended with red post-mortem clots. Valves normal. Just above valvular insertion aorta shows diffuse areas of fatty atheroma. Both coronaries normal. Consistency of the myocardium firm; on section of a homogeneous red-brown color.

*Left Lung*.—Free from adhesions. Both layers of the pleura smooth. Consistency firmer than normal. On section all lobes present a brownish-red color and there escapes a considerable quantity of blood-stained serum. No thickening at the apex.

*Right Lung*.—Lower lobe adherent by fresh adhesions. Both layers smooth except where adherent over lower lobe; here the pleura is covered by lymph and fibrin. Upper lobe on section contains a small amount of serum. Lower lobe is perfectly solid to the feel. On section the hepatized lung presents a yellowish-gray color, with streaks of brown or black. Surface somewhat finely granular and lung contains practically no fluid. Bronchi: mucous membrane congested on both sides. Blood-vessels normal. No apical tuberculosis. Bronchial glands slightly enlarged and pigmented; free from calcareous deposits or caseation.

*Liver*.—Free from adhesions; not enlarged; consistency



soft. Capsule smooth and on section yellowish-brown or red color. Very cloudy.

*Gall Bladder*.—Normal. Common duct patent.

*Spleen*.—Free from adhesions; moderately enlarged; consistency firm (due to hardening fluid). Capsule smooth; trabeculæ not increased; malphigian bodies swollen; pulp increased.

*Left Kidney*.—Fatty capsule small in amount. Fibrous capsule strips off easily. Surface smooth. On section cortex normal in amount; cortex markings distinct; glomeruli visible. Medulla and pelvis normal. The *right kidney* presents a similar appearance to its fellow.

*Ureters*.—Both normal. *Pancreas* likewise normal.

*Left Adrenal Gland*.—Enlarged, soft, and on section contains a pea-sized cavity, walls of which are of a dark brown color and caseous. Contains a small quantity of dark brownish-red fluid; viscid consistency.

*Right Adrenal Gland*.—Also enlarged; somewhat firmer than normal; on section shows a number of diffuse areas of yellow suggestive of caseous material. These areas are firmer, however, than caseous material generally, due to calcareous deposits.

*Aorta, Bladder, Stomach, Æsophagus, and Prostate*.—Normal.

*Mesenteric Glands*.—Enlarged, and on section caseous; the retroperitoneal glands are swollen to twice their usual size. No distinct caseous areas can be made out.

*Tongue, Epiglottis, Vocal Cords, and Larynx* present a normal appearance. Trachea moderately congested. Thyroid normal.

*Intestines*.—These are free from adhesions. As one passes from the duodenum and jejunum to the ileum, noticeable increase in the thickness of the wall becomes apparent in the upper third of the ileum, but the change from the normal to the thickened ileum is scarcely



perceptible. Where the change occurs the intestinal wall feels harder, more stiff, until finally when the middle third of the ileum is reached, the gut occurs as a cylindrical tube, with non-collapsible walls of firm, hard, resistant tissue. At a point corresponding about to the junction of the upper and middle thirds of the ileum, the wall has a thickness measuring from 4 to 7 mm. Passing downward, the thickening rapidly becomes more evident, especially in the last half metre of the small bowel, where the wall measures 1 cm. in thickness. The cæcum is thickened also and bound down laterally to the abdominal wall by old fibrous adhesions. The thickness of the wall here measures 2.7 cm. The ileo-colic valve is thickened and rather rigid. No loss of substance can be made out. The ascending colon presents a similar appearance to the other thickened intestine, but measuring not over 1.2 cm. near the cæcal end, and at the hepatic extremity only .75 cm. The transverse colon measures about the same (.75 cm.); finally, the descending colon imperceptibly diminishes in thickness to the sigmoid flexure, when the bowel again assumes its normal proportions. The rectum is normal.

On section the thickened portions are resistant to the knife, and gray or whitish in color. The thickening is quite uniform in all portions of the circumference of the intestine, except in the cæcum, where the thickening is most pronounced posteriorly; here the thickness is fully 1 cm. greater than anteriorly. To the naked eye the walls seem to be largely fibrous. No tubercles or caseous areas can be made out. When opened the lumen is seen to be perfectly patent at all points. It is quite normal in the ileum above, but in the lower half-metre it appears to be reduced by about one third. The same holds good for the remainder of the intestine until the transverse colon is reached, where the diameter gradually becomes more and

more normal as the sigmoid is approached. The cæcum is free and the circumference of the lumen is 13.4 cm.; that of the ileum, one half-metre above the ileo-cæcal valve, 7 cm.; in the ascending colon, 9 cm.

The mucous membranes of the duodenum and jejunum are bile-stained, but smooth and normal in appearance. In the ileum the mucous membrane seems pale, and everywhere free from apparent ulceration or evidences of such. The same holds good for the remaining length of the canal. Now and again swollen solitary follicles may be seen in the small and large intestine; Peyer's patches appear unaffected. Beginning in the middle of the ileum, the mucosa becomes thicker and thicker until the ileo-cæcal region is reached; in the cæcum and ascending colon it appears about the same and therefrom becomes more and more normal. The most conspicuous feature of the mucosa consists, however, in the presence of numerous papillomatous masses, most abundant in the lower ileum, and having an average size of about 4 or 5 mm.

The peritoneal surface is everywhere smooth and no evidence of old peritonitis is to be found; nowhere can tubercles be detected.

#### *Microscopical Examination.*

The histological examination of the various organs confirmed in the main the findings at the autopsy table.

*Intestine.*—The mucosa was much thickened, about four or five times beyond the normal. This thickening was largely due to massive infiltration with round cells. The surface epithelium looked normal. The papillomatous outgrowths were directly continuous with the submucosa and present some general round cell infiltration observed throughout the various layers. They further contain unstriated muscle fibre continuous with the muscularis mucosa. The submucosa presented the greatest degree of



round-cell hyperplasia; greater or less similar infiltration also existed in the muscular zones. No histological tubercles were observed, but sections stained for tubercle bacilli revealed great numbers, especially in the mucosa and submucosa.

---

*Stated Meeting, April 11, 1900.*

EUGENE HODENPYL, M.D., PRESIDENT.

A PRONOUNCED CASE OF ARTERIO-SCLEROSIS.

Dr. D. H. McALPIN, Jr., presented specimens from a patient who had been admitted to hospital on March 19th, suffering from dyspnœa and pain in the epigastrium. He was fifty-four years of age, and had been a sailor until a short time ago, when he had been compelled to give up this occupation because of shortness of breath. His past history was not a good one, having had syphilis and gonorrhœa, and having been accustomed to indulge in alcoholics to excess. Ten years previously he had fallen from a height and had fractured his clavicle. The present illness had begun with pain in the stomach and continued vomiting, shortness of breath, and some cough. Shortly after admission he had become delirious and had remained so until death. A few hours before death there had been complete suppression of urine. His temperature had ranged between 100° and 102° F.; the pulse had been 80, and the respirations between 28 and 40. There had been signs of consolidation of the lungs, and the apex of the heart was at the sixth interspace, to the left of the nipple line. There was a murmur heard indicating aortic insufficiency. The diagnosis had been chronic endocarditis and nephritis, pneumonia or possibly infarctions of the lungs, and arterio-sclerosis. At the autopsy, on opening the chest the heart had been found greatly enlarged.



It weighed nearly fifty ounces. All of its cavities were greatly distended, and contained large, dark clots, and the coronary vessels stood out very prominently but were not tortuous. On opening the heart, several thrombi were found at the apex of the left ventricle, and a number of thrombi were attached behind the columnæ carneæ. The mitral orifice admitted four fingers, yet the valve curtains were greatly enlarged and appeared to be perfectly competent. The aortic orifice was very large, and the curtains of the valve were also very large and free. There was calcification of the aorta beyond the aortic valve, but no visible disease of the curtains. The walls were considerably hypertrophied, and the cavities greatly dilated. On the right side there appeared to be no disease of the valves. The foramen ovale was patent. The liver was about normal in size, and on section it showed a distinctly nutmeg character. There was an immense amount of passive congestion in the central zone of the lobule. The small hepatic arteries and hepatic veins were very thick and easily seen. The kidneys were of characteristic stony hardness of the cardiac kidney. The small arteries were very prominent. The lungs exhibited areas which were firm, more or less granular, and of a gray color. The vessel walls were greatly thickened. The same uniform thickening of the vessels was observed in the brain.

#### A CASE OF PERFORATION OF THE STOMACH BY A TOOTHPICK.

Dr. McALPIN also presented the case of a man, aged fifty years, a chronic alcoholic. Four months before admission he had been hit in the epigastrium with a baseball. Two months later his abdomen had begun to swell, and he had then begun to lose flesh very perceptibly. His temperature on admission had been 100° F., but had quickly fallen to normal. The abdomen had been found

distended with fluid; the heart was normal; the lungs were the seat of emphysema. On February 15th he had been so much better that he had left the hospital for the day, and had not returned until March 10th. At that time he had been jaundiced and feverish, and his urine showed hyaline and granular casts. He had died on March 14th, three hours after one gallon of dark, foul-smelling fluid had been removed from his abdomen. This fluid contained a variety of germs and leucocytes. At the autopsy, the intestines had been found greatly distended and heavily coated with fibrin, and the omentum hemorrhagic and drawn over the liver. The starting-point of the peritonitis had been traced to a point underneath the liver. On separating the adhesions here, a cavity one and a half inches in diameter had been laid open. This cavity was formed by the stomach and the liver. It contained dark, greenish viscid pus. In this cavity, sticking into the stomach, was a piece of a wooden toothpick, one inch in length. The opening through which the wooden spicula had entered the peritoneal cavity could not be found, evidently having closed by muscular contraction as soon as the wood had passed.

#### A SPECIMEN OF UTERINE MOLE.

Dr. F. R. BAILEY presented this specimen, which had been removed from a colored woman, about fifty-five years of age. She had been married about five years, and had had no children and no miscarriages. Her last menstruation had been on July 17, 1899. On November 29th she had had a severe hemorrhage, and on February 1, 1900, she had had what she called a miscarriage, *i. e.*, a discharge of a foetus of five or six weeks embedded in a mass of tissue, for the most part placental.

## THE DURATION OF LIFE OF TYPHOID BACILLI IN ICE.

Dr. W. H. PARK said that he had been making some experiments on the duration of life of typhoid bacilli in ice. He had been led to do this because of some statements made recently by Dr. Sedgwick as to the rapidity with which death occurred. He had experimented with twenty cultures. They had been made from twenty-four-hour agar cultures, one loop of each culture being put in a separate lot of 300 c.c. of filtered Croton water. The average number of bacilli in the water of the twenty lots was 2,500,000 in 1 c.c. At the end of three days' freezing the average number was 1,089,000; at the end of one week, 361,000; at the end of two weeks, 203,000; at the end of three weeks, 10,000; at the end of four weeks, 4000; at the end of five weeks, 3000; at the end of seven weeks, 2000; at the end of nine weeks, 127. Three of the cultures had become sterile at this time. In general, then, it might be said that at the end of nine weeks the ice from a certain proportion of the cultures was sterile, and the living bacilli from all the cultures were greatly reduced in number.

*Discussion.*

Dr. T. M. PRUDDEN asked if any cultures older than twenty-four hours had been tested. He asked this because he thought it not improbable that at this age the bacilli were more vulnerable than older ones.

Dr. PARK replied that the cultures differed greatly as to the lapse of time since they were obtained from the original source. Of some interest was the fact that in thirty specimens of ice taken at random neither colon nor typhoid bacilli had been found, though the ice was the ordinary ice found in the market.



Dr. A. J. LARTIGAU said that last year he had had occasion to investigate the ice supply of the city of Albany. From one specimen he had obtained an organism which responded to all of the culture tests for the typhoid bacillus. Unfortunately this specimen had been destroyed before it could be tested with typhoid serum. There was every reason to believe that this ice was over three months old. He had tested pure cultures of typhoid bacilli at various low temperatures. At the end of twenty-four hours nearly fifty per cent. had been destroyed—in some specimens a still higher percentage were killed—and at the end of ten to eighteen days practically all had been destroyed (ninety-seven to ninety-eight per cent.). The water used was the Hudson River water. He had also tested the viability of the bacilli in water just above the freezing-point, and had found this temperature about as effective as when the water had been frozen. Alternate freezing and thawing had been somewhat more destructive to the life of the bacilli.

#### A PECULIAR STREPTOCOCCUS.

Dr. E. LIBMAN presented a streptococcus which had been isolated from the stools of a child suffering from acute enterocolitis. The stools had been placed in sugar bouillon for twenty-four hours, and this had yielded a pure culture of this organism. This streptococcus was peculiar especially in one respect. In its growth on lactose agar and glucose agar it made the medium white, which it did not do in its growth on the other preparations of agar. The peculiar action was found to be due to a precipitation of the albumin in the medium by the acid formed by the splitting up of the sugar. The organism was pathogenic for mice.

Dr. LIBMAN then read a paper entitled

## SARCOMA OF THE SMALL INTESTINE.

During the past two years there have been observed at the Mount Sinai Hospital five cases of intestinal sarcoma, four of which came to autopsy. In three of these cases the clinical picture closely resembled that of appendicitis. In attempting to ascertain whether such a resemblance had before been noted, I found that not only was the clinical picture unknown, but also that there had been no complete review of the subject of sarcoma of the intestine since Baltzer wrote his article in 1893.

In the following paper I shall give the details of the four cases of ours which came to autopsy, and refer occasionally to the fifth case. I shall then attempt to give a complete picture of the disease from all its standpoints. I am indebted to Dr. Gerster for his kindness in permitting me to observe four of these cases clinically, to Dr. Lilienthal, in whose service the fourth case occurred, and to Dr. Brewer, by whom it was operated upon.

CASE I.—H. G., aged twelve years, schoolboy, admitted September 30, 1898. History is of nine days' duration. The boy first complained of pain in the abdomen, below and to the right of the umbilicus, and of weakness. Three days ago abdominal distention was noticed, and since then there is marked constipation. The distention and pain have increased, and the boy suffers from dyspnoea.

On admission the temperature was 100.6°. The status in short was the following: Superficial abdominal and thoracic veins distended. Liver: upper border of dulness at fifth space, lower border undetermined. Abdomen much distended, signs of free fluid. Across the hypogastric region and extending into the pelvis there is a nodular tumor, concave above. Rectal examination revealed a large bulging mass anteriorly.

Operation, on October 1st, by Dr. Van Arsdale. The



peritoneum was found lined with white nodular masses, and a large amount of chyliform fluid escaped. After the operation the boy gradually became weaker, there developed a left-sided empyema, purulent peritonitis, and otitis media. He died on October 29th.

*Post-mortem Examination.* (Abstract.) Lungs: large purulent exudate in left pleura. Small abscess in base of left lung. Infiltration of the diaphragm and base of the left lung by new growth. Evidences of healed tuberculosis in the right lung. Spleen: infiltrations on surface. Kidneys: in the medullary rays brown streaks looking like uric acid infarcts; growths on anterior surface. Pancreas: diffusely infiltrated. Liver: size normal; fatty; covered by flat growths, white in color, some of which involve the parietal peritoneum. The wall of the gall bladder is uniformly infiltrated. The omentum and mesentery are very much thickened, due to infiltration by tumor masses. The mesenteric nodes are all very large, and on section are white, homogeneous, and dry. Stomach: walls diffusely infiltrated. Intestinal coils are bound together by masses between which are sacculations of green pus. The parietal peritoneum is not involved, except over the liver. The intestinal coils are attached to the bladder by some of the growths. The walls of the colon and part of the ileum show a thin infiltration with the new growth, the mucosa not being involved. In the duodenum, eight centimetres from the pylorus, is a large growth which encircles the gut, and projects into its lumen. The growth measures seven centimetres in length, five in width, and six in thickness. The mucosa is intact. Microscopical examination: lymphosarcoma. (Some of the details of the microscopical findings will be given later.)

NOTES. In this case of sarcoma of the duodenum, with extensive metastases, there existed stenosis of the



intestine, which, as we shall later see, is the exception to the rule. The case was sent into the hospital with the diagnosis of "appendicitis." The diagnosis made in the hospital was "new growth of the peritoneum, probably sarcoma."

CASE II.—A. R., aged three and one half years, admitted December 23, 1898. History of eight days' standing. The child complained of abdominal pain, and the abdomen was seen to be enlarged and rigid. Bowels moved freely, but there was difficulty in urination. During the last three days the child vomited several times, and was feverish. The feet were not swollen.

The physical examination revealed the following: The liver flatness begins at the sixth rib, and extends four centimetres below the free border. Spleen is enlarged to percussion. The right side of the abdomen is occupied by an irregularly shaped mass extending to the median line, easily pushed about, moving freely with respiration. The lower limit seems to be at the umbilicus. The surface feels irregular, as if the mass were composed of lymph nodes. The abdomen is symmetrically distended. There are some hard, round masses to be felt just above the groins. Temperature 100.8°.

25th. Urine contains considerable indican. Catheterization found necessary. Blood examination shows a moderate secondary anæmia and moderate polynuclear leucocytosis.

31st. Abdominal distention more marked. Intense dyspnœa.

January 5th. Operation by Dr. Gerster. Peritoneum found very much thickened. Some ascitic fluid escaped. The intestines were found adherent to each other, and to the omentum. The patient died on the following day.

*Post-mortem examination* showed the following changes: Lungs: large effusion of clear fluid in the left pleura.

Abdomen: very much distended. The mesentery and omentum are everywhere thickened and whitish. All the intra-abdominal lymph nodes are enlarged, some being four centimetres in diameter. On section they are white, succulent, soft. In the beginning of the ileum there is a large, white tumor infiltrating the wall, especially opposite the mesenteric attachment. The main tumor mass measures seven by eight centimetres, and is about three centimetres thick. On opening the intestine the growth is found to be indurated, and has an elevated irregular edge. Except for a few small areas of necrosis the mucous membrane is intact. The part of the ileum involved is considerably dilated, the circumference there being three times as great as that of the uninvolved part of the intestine. There is also an infiltration of the wall of many of the intestinal coils, and the coils are adherent to each other. The iliac lymph-nodes are very much enlarged and adherent to the bladder-wall, which is much thickened and nodular. The ureters are partially obstructed by the tumor. The parietal peritoneum is involved, but to a lesser degree than the mesentery and omentum. Spleen: moderately enlarged; few nodules on the surface; slight infiltration along the vessels coming from the hilus. Pancreas: entirely infiltrated and very much enlarged. Kidneys: normal in size; cut surface yellowish. In the left kidney there are a few small nodules. The pelves of both kidneys are somewhat dilated, the left more than the right. Left adrenal entirely replaced by new growth, and adherent to the pancreas. Stomach: wall whitish and thickened. Liver: very much enlarged. Almost the whole right lobe is light brown in color, and is separated from the normal liver tissue by a raised edge. This lighter part is hard on section, and appears to be uniformly infiltrated by new growth. There are also a number of small, hard nodules present. The liver as a



whole is somewhat fatty. Gall bladder: distended with a colorless mucoid fluid; the neck of this organ is much thickened by new growth, and the cystic duct is impassable. Appendix is eleven centimetres long; the wall is much thickened and uniformly infiltrated with new growth. Bronchial nodes present the same appearance as the intra-abdominal nodes. Microscopical examination: lymphosarcoma.

NOTES. This second case is an instance of a primary intestinal sarcoma with dilatation, and with the most extensive metastases yet reported. The diagnosis before operation wavered between sarcoma of the kidney, tubercular peritonitis, and sarcoma of the peritoneum, with a possible primary intestinal tumor. The clinical history, except for the bladder symptoms, was rather typical.

CASE III.—M. G., aged eighteen years; Russia; admitted January 22, 1899. History is of one day's standing; began with abdominal pains, most marked on the right side below the umbilicus. Bowels moved yesterday. Has been vomiting since last night, vomitus being bile-colored. No history of any previous attacks. Status: Hippocratic appearance; breathing rapid and shallow; pulse almost imperceptible; legs drawn up; abdomen tense and hard, uniformly tympanitic. By rectum a large doughy mass is felt high up. Operation by Dr. Van Arsdale. Saline infusion. The abdomen was opened in the right iliac fossa, and a large amount of fluid, fecal matter, and serum poured out. The appendix was found normal. The abdomen was then opened on the left side, and the peritoneal cavity irrigated.

Temperature on admission  $103.6^{\circ}$ , respirations 50, pulse 100, almost imperceptible. Freely stimulated. After operation pulse rapidly gave out.

The *autopsy* showed the following: In the jejunum,



one and one third metres from the duodenum, is a perforation in the intestine measuring seven by eight centimetres in diameter, with an irregular edge. The perforation is due to a tumor which has infiltrated the wall of the intestine and dilated it. The growth is white and soft on section, and varies from one half to two centimetres in thickness. The tumor surrounds the circumference of the intestine, except for a space of three centimetres on the mesenteric side. Just above the tumor in the mesentery anteriorly is a node which looks cheesy. Posteriorly there is a lymph node infiltrated with sarcoma. There were no metastases found.

Microscopical examination of the tumor and the infiltrated nodes shows lymphosarcoma.

NOTES. This unique case is an instance of intestinal sarcoma which produced no marked symptoms until perforation occurred. The diagnosis of general peritonitis, probably due to perforation of the appendix, had been made.

CASE IV.—M. M., aged forty-two years, admitted January 8, 1900. For the last two weeks the patient has had irregular abdominal pain. During the past week he has suffered from frequent urination. No blood was passed, nor was there a urethral discharge present. Four days ago he was seized with severe abdominal pain, especially marked in the pelvis. There has been absence of fever, chills, and vomiting. The bowels were constipated until yesterday, although cathartics and enemata had been used. Within the last four days the patient has noticed a mass low down in the abdomen, which has grown rapidly and has become very tender.

The physical examination of the emaciated patient revealed an irregular tumor in the hypogastric region, extending into both iliac fossæ, especially the right. The tumor is quite tender, rather hard, but gives the sense of

deep fluctuation. The rectal examination shows a hard symmetrical bulging. The temperature was  $101.4^{\circ}$ ; the urine contained albumin, pus cells, and hyaline and pus casts.

Operation on day of admission by Dr. Brewer. On opening the abdomen a very large hemorrhagic tumor was found springing from the ileum and adhering to the right iliac wall, the floor of the pelvis, the large vessels, and the bladder. After much difficulty the tumor, together with two inches of the small intestine, was removed, and a Murphy button anastomosis performed. The patient died three days later from acute peritonitis.

The description of the tumor is as follows: It springs from the ileum at a distance of seventy centimetres from the valve. At its point of attachment to the intestine there is a beginning diverticulum. The tumor is very irregular in shape, and consists in general of two more or less reniform parts. The greatest width of the tumor is fifteen centimetres, the length thirteen centimetres, and the thickness eight centimetres. One half of the growth is entirely hemorrhagic and cystic, and the other half is necrotic and cystic. On cutting the tumor open it is found to begin in the submucosa, reaching to the mucosa, but not involving it. Microscopical examination: spindle-celled sarcoma.

Of the *autopsy* notes only the following are of special interest: The spleen shows acute inflammation and hemorrhages. The kidneys show chronic nephritis, with acute degeneration. In the liver there is a marked pigmentation, presumably due to absorption of blood from the tumor. There are no metastases present.

NOTE. This case was one of a solitary tumor of the intestine, which clinically bore the closest resemblance to acute appendicitis, the acuteness of the symptoms being due in all probability to the hemorrhagic extravasation.



ETIOLOGY. *Frequency.* A glance at some statistics will be necessary to show the frequency of sarcoma of the intestine, and the comparative frequency of sarcoma and carcinoma. From 1859 to 1875 there was no case of intestinal sarcoma observed in the Berlin Pathological Institute. On the other hand, Smoler reports thirteen cases in Prague in fifteen years among 13,036 autopsies. Nothnagel states that in twelve years there came to autopsy in Vienna twelve cases of intestinal sarcoma. This certainly indicates that the disease is an unusual one. Possibly the occurrence of four cases of lymphosarcoma in our service in a very short time would tend to show that we may have to deal with an endemic disease, for, as we shall later see, there is reason to suspect these tumors to be of infectious origin.

Compared to the total number of lymphosarcomata the intestinal cases are not very infrequent. Nothnagel's figures show that among 274 sarcomata three involved the intestine, and of sixty-one lymphosarcomata nine were primary in the intestine.

As to the frequency compared to carcinoma, Mueller reports 521 cases of carcinoma, of which forty-one occurred in the intestine, and 102 cases of sarcoma, with only one instance of intestinal involvement. Similarly, Nothnagel's figures show that among 2125 carcinomata 243 occurred in the intestine; of 274 sarcomata three occurred in the intestine, and among sixty-one lymphosarcomata there were nine of the intestine. So that sarcoma of the intestine is much more uncommon than carcinoma.

*Location.* Sarcomata have their seat of preference in the small intestine. In the large intestine they are much more uncommon, except in the rectum, where they occur quite as frequently as in the small intestine. A few figures will illustrate this. Krueger gives the following statis-



tics of thirty-eight cases: Small intestine, sixteen; ileum and cæcum one; cæcum, two; appendix, one; transverse colon, one; small and large intestine, one; rectum, sixteen. Nothnagel's figures are as follows: Of nine lymphosarcomata one involved the duodenum; three the jejunum; three the ileum, and two the cæcum. Of three sarcomata one occurred in the ileum; one in the cæcum, and one in the rectum.

*Age.* Baltzer states that fifty-eight per cent. of the cases occur in the fourth decade, and in eleven of his fourteen cases the age was not over forty. I have collected fifty-one cases in which the age is noted. The results are as follows: First decade, six; second, nine; third, thirteen; fourth, thirteen; fifth, eight; sixth, one, and seventh, one. This shows that the age cannot be used as a diagnostic point. The oldest patient was seventy (Smoler). The youngest was a congenital case reported by Stern. This case was that of a child which died when five days old with symptoms of intestinal obstruction. There was found at autopsy a round-celled angiosarcoma of the jejunum.

*Sex.* Baltzer's cases, with one exception, were all males. I have collected forty-nine cases including Baltzer's. Among these we find thirty-five males and fourteen females; or, in other words, the disease seems to be more than twice as common in males as in females.

*Cause.* As to the causation but little is known. In several instances the disease developed after a trauma, the usual interval until these symptoms developed being five to six weeks (Jalland, Pepin). In one case there is a previous history of syphilis. In another (Nothnagel) it followed a tuberculosis of the intestine. Flexner, in reporting two cases, drew attention to a histological feature of the growth, which he believed might throw a hint as to the cause of lymphosarcoma. He found in the growth

in the stomach, intestine, and kidneys (most distinctly in the last) certain peculiar bodies. These were oval, round, or slightly irregular in shape, and consisted of a rim of protoplasm staining faintly with eosin, and enclosing a particle staining with hæmatoxylin. The last was oval or crescentic, and lay either in the centre of the cell or eccentrically. The bodies were distributed irregularly in the diseased areas, and an occasional body was seen in the adjacent parts. Flexner believed that they were probably protozoa, but stated that they might have no causal relationship.

I have found these bodies in the sections from the involved organs in our cases of intestinal lymphosarcoma, and also in specimens from other cases of lymphosarcoma. Although in places they at first impress one as probably being fragments of cells disintegrated by the infiltration of the new growth, a careful examination shows that such a view is not tenable, and one must agree with Flexner's view that they are at least suspicious.

**PATHOLOGY.** *Location and Size.* I have collected fifty-two cases in which the location is given. Of these, fifteen occurred in the duodenum; eighteen in the jejunum; two involved the jejunum and ileum; fourteen the ileum, and three the entire intestinal tract. These figures show that the larger number of sarcomata occur in the jejunum and ileum, but they may be located anywhere. The tumor may be single or multiple, small or large.

*Varieties.* The varieties of tumor found are: spindle-celled sarcoma, lymphosarcoma, myosarcoma, endothelioma, interfasciculare, round-celled sarcoma, melanosarcoma, and mixed-celled sarcoma. Some round-celled sarcomata might better be classed with the lymphosarcomata.

When sarcoma occurs in the intestine it is generally



primary there. It may, however, be secondary, or it may be part of a general lymphosarcomatosis. In this article we shall direct our attention to the primary cases only. In most of the cases the growth is confined to the mucosa and muscularis, and the serous coat is entirely or nearly entirely free. In a very few instances the growth began in the serosa, and later involved the inner coats. The lymphosarcomata, which formed the largest group (seventeen cases), generally begin in the submucous lymphatic nodules, and have a tendency to grow longitudinally. The muscularis is early infiltrated and paralyzed, and the feces dilate the intestine. This dilatation is a peculiar though not a constant feature of intestinal lymphosarcoma.

**METASTASES.** I have arranged these according to the character of the growth. Among the cases reported with microscopical examination there are five instances of spindle-celled sarcoma. Four of these had no metastases; one had metastases in the peritoneum and liver. The location of the tumor is not always given, but one was in the ileum, and our case was also in the ileum. Of the lymphosarcomata (seventeen cases) three involved the duodenum; four the jejunum; three the ileum; one appendix; six the ileum and jejunum, and one the whole intestinal canal. Metastases occurred as follows: In three cases there were none; in most of the cases the mesenteric nodes and other parts of the intestinal walls contained growths. The liver was involved seven times; the kidney seven times; the spleen three times; the pancreas once; the adrenals once; the diaphragm twice; the rectal wall twice, and the bladder four times.

Among the numerous other reported cases we will give details of a few only. In the case of Nicolaysen (myosarcoma) a few nodes in the mesentery were involved, and in Lehmann's case of endothelioma the whole



intestine except twelve inches was involved. Of the round-celled cases (four) two involved the jejunum and two the ileum. In one case there were no metastases; in another there were growths in the mesentery, and in the third the kidneys and upper part of the rectum were infiltrated. In Treves' case of melanosarcoma of the ileum the inguinal glands were infiltrated.

These data all go to show that the spindle-celled sarcomata have few or no metastases. The lymphosarcomata generally have extensive metastases, every abdominal organ and tissue being liable to invasion. The superficial lymph nodes are not generally involved. The metastases occur most commonly in the peritoneum, lymph nodes, liver, and kidney. There is a marked tendency for the growth to occur in the pelvis or for the original tumor to become adherent there. In our first two cases the growths seem to have been more extensive than in any other cases hitherto reported. A careful examination of the specimens showed that the growth extended almost entirely by continuity, or by contact. In the liver the growth extended inward from the surface, or along the vessels. The spleen and kidneys showed the same.

The parietal peritoneum was implicated only by extension from the visceral peritoneum. This opposes the statements made about the discontinuous growths of the lymphosarcomata, and strengthens the impression that these growths are infectious in nature, and that we are not dealing with metastases in the true sense of the word.

**MICROSCOPICAL EXAMINATION.** I shall confine myself here to a few notes of my own cases. The fourth case needs but little commentary. In the other three cases the picture was that of a typical lymphosarcoma. I might mention, however, the great number of large lymphatic capillaries in some of the growths, and the frequent

occurrence of the growth in the lumina of these capillaries.

In the first case the main growth was found in the submucosa, and through slits in the muscularis mucosæ the sarcoma cells reached up between the tubules. In some places the muscularis was entirely replaced by new growth. In the mesentery, omentum, and intestinal wall the tumor seemed to pass along under the serosa. The mucosa of the intestine above and below the growth showed no particular changes. The liver showed acute degeneration and congestion. The infiltrations in it followed the vessels, and tended to surround the lobules. There was some increase of the connective tissue between the lobules in the parts of the organ which were not infiltrated. The capsule was distinctly thickened. In the kidneys there was present a marked degeneration and congestion.

In the second case the sarcoma cells could very frequently be seen in the lymphatics. The liver showed fatty degeneration and infiltration and acute congestion. The growth seemed to spread between the liver cells, and at the edge of the infiltration the liver cells were almost entirely replaced by fat, or appeared to be necrotic. Near the growth the blood-vessels were much dilated. At a distance from the growth there was a marked parenchymatous degeneration, but less fatty infiltration. In one place there were small calcific deposits. The spleen was congested and hemorrhagic. The infiltration spread here also along the connective tissue and around the vessels, and there could frequently be seen an infiltration about the vessels in the Malpighian bodies. In the kidneys there was found an acute congestion and degeneration. The infiltration occurred between the tubules, and in the areas involved the tubular epithelium was very indistinct. The stomach was diffusely infiltrated, even the mucosa being involved. In the gall bladder the infiltration



involved the submucosa only. The mucosa of the intestine above and below the primary growth for a short distance stained homogeneously and showed no distinct structure.

In the third case there was a cheesy gland in the mesentery, but microscopically this proved to be lymphosarcomatous in nature. In the fourth case we will note only that the liver was markedly pigmented, that the kidney showed an acute parenchymatous degeneration, that the spleen was acutely inflamed and hemorrhagic, and that the heart presented a brown atrophy.

I have given the microscopical report of these cases to bring out the following points:

1. The transmission alongside the vessels, and the growth in the lymphatics in the lymphosarcoma cases.

2. The marked degeneration in those portions of the liver next to the new growth. Schulz (*Archiv für Heilkunde*, Band xv., p. 193) cites several instances of lymphosarcoma of the liver, stomach, intestines, and kidneys in which the epithelium near the growths was swollen, very granular, often fatty and disintegrated.

3. The parenchymatous degeneration of the liver and kidneys as a whole. In our second and fourth cases this must be explained as being due to the absorption of toxins or allied substances from the growths, and this again points to their probable infectious nature.

4. The fact that a cheesy gland may be lymphosarcomatous (*infra*).

5. The brown atrophy of the heart in the last case indicates a long continuance of the growth before the occurrence of distinct symptoms.

Flexner noted in his two cases extensive atrophy of the mucosa of the entire intestinal tract. In our case no such change was present, except to the slight degree noted in the second case.



RESULTS OF THE GROWTH. (a) *Dilatation and Stenosis.* As pointed out by Kundrat, Treves, and Baltzer the lymphosarcomata cause dilatation of the intestine. This may be aneurismatic in form, as in the cases of Haas, Bessel-Hagen, and in our second case. Occasionally the tumor causes stenosis, but there is no case on record of complete stenosis due to obturation of the lumen by the tumor. Rutherford stated that the higher up the tumor lies the less likely is there to be stenosis. But we have noted that the stenoses have been found most commonly in the sarcomata of the duodenum. When a complete obstruction has been found it has been due to one of the following causes: (1) Invagination (Wallenberg); (2) twisting of the mesentery, due to the tumor being caught in a hernial sac (Waldenström); (3) adhesions (Schmidt). The tumor may be attached to the intestine by a pedicle, as in Lannelongue's case. In our fourth case the tumor had produced a small diverticulum in the wall by traction.

(b) *Results of Compression.* The tumor may compress:

1. The vena cava, causing œdema of the legs and ascites.

2. The bile ducts, and pancreatic duct (Lancereaux, sarcoma of the duodenum). In our second case the cystic duct was obstructed.

3. Ureters. This occurred in our second case, and resulted in a hydronephrosis of a moderate grade.

(c) *Ulceration and Perforation.* Ulceration is quite common, and may result in perforation, such perforation being either closed off by intestinal coils, the bladder or rectum, or opening directly into the general peritoneal cavity. The perforation may occur in any part of the intestine. The ulceration may expose vessels (Rolleston, sarcoma of the duodenum, erosion of the inferior pancreatico-duodenal artery). It is possible for these tumors to rupture without ulceration (Zuralski).

(d) *Changes in Other Parts of the Body.* The general changes and metastases have been described above. The pleural cavities may contain clear fluid or pus. There may be a localized or general peritonitis, the effused fluid being chyliform, clear, purulent, or hemorrhagic. There are practically no reports of a careful examination of the effusions in these cases. In the case reported by Henoeh the fluid was hemorrhagic and contained numerous round cells showing fatty degeneration. We shall later give the details of the fluid in our fifth case.

(e) *Connection with Other Diseases.* In one case there was found a mixed-celled sarcoma of the ileum and an adenocarcinoma of the pylorus (Smoler). More important is the fact that lymphosarcoma has frequently been found in persons having either an intra-abdominal tuberculosis or a florid or healed tuberculosis elsewhere. The exact connection is not understood. While not attempting to state that a combination of tuberculosis and lymphosarcoma is not frequent, we believe that only cases in which a microscopical examination is reported should be credited, for just as Sternberg has shown that a lymph node that looks lymphosarcomatous macroscopically may be tubercular on microscopical examination, so I have shown that the opposite mistake can be made.

**SYMPTOMATOLOGY.** *General Description.* Nothnagel gives us a good outline of the disease when he states that there is very early and without exception a marked affection of the general system and but few local symptoms. Baltzer's description, which has been quoted in most of the books, is as follows (abstract): "The symptoms are very slight at the onset. There is at first pain in the stomach, loss of appetite, nausea, vomiting; the bowels are irregular, being either constipated or loose. The abdomen soon becomes distended. When seen early all the patients are very thin and have a pale color. A tumor is



generally found, although it may be missed for a long time; it may be located in any part of the abdomen, and it is generally but slightly or not at all tender. The temperature is normal or moderately elevated. In some cases there is a leucocytosis. The duration of the disease varies from two weeks to one and three-quarter years, but most of the patients died within nine months."

DETAILED SYMPTOMATOLOGY. *Pain* is present in practically all the cases. It may be mild or severe, and may be located in any part of the abdomen. In most cases the pain is referred to the stomach, and occasionally it is especially marked after meals. In three of our cases the pain was specially referred to the region of the appendix.

*Distention of the Abdomen* is very common, being uniform or irregular. Its onset is frequently sudden, and the symptoms of the disease may date from its appearance. The swelling may rapidly increase or remain at a standstill, later to become suddenly more marked. The distention is due either to involvement of the peritoneum, pressure on the vessels, perforation of the intestine, tympanites, or the size of the tumor. If fluid is present it may be clear, chyliform, purulent, or hemorrhagic. In our first case it was chyliform, in the second clear, and in the third fecal. In the fifth case a paracentesis abdominis was made, and the fluid showed the following: It was cloudy, yellow, odorless; its specific gravity 1.014. There was present a trace of urea; albumin, 0.12 (.1290) per cent. Microscopically there were found a large number of pus-cells, red blood-cells, and some epithelial cells, numerous cocci and bacilli. Culture showed the bacterium coli commune.

CHARACTERISTICS OF THE TUMOR. In some cases a tumor cannot be made out. If a tumor is discovered it may be first found by examination, or it may be the



symptom which leads the patient to consult a physician. After the tumor is once there it may grow very rapidly. The growth may be large or small, single or multiple. The lymphosarcomata often feel like one irregular tumor, whereas in reality the growth in the intestine is very small, and the irregularity is due to growths in the peritoneum and lymph nodes. At times small nodules are found which feel like lymph nodes, but which on post-mortem examination prove to be infiltrated appendices epiploicæ. The tumor may be superficial or deep. It may be possible to feel one large tumor and scattered nodules all over the abdomen, some feeling as if they were in the parietal peritoneum. Such an observation was made in our second case.

The tumor is generally only slightly or not at all tender. Marked tenderness occurred in only one of our five cases. The growths usually move with respiration, and can generally be moved around in the abdomen. The note over them is generally dull or dull tympanitic. Their consistency is generally moderately hard, although very soft and very hard ones have been described. The centre may show indistinct fluctuation, as in our fourth case. Crepitation may be felt over the tumor, and the latter may be seen to take part in the peristaltic movements of the intestine.

The position of the mass is most variable, but a large number of them tend to grow downward, or the growth occurs primarily in the lower part of the abdomen. As a result a rectal examination may reveal a tumor when nothing or only small nodules can be felt in the abdomen. In three of our cases parts of the growth could be felt by the rectum.

*Symptoms Due to Compression.* These may be summarized as follows: 1. Ascites or œdema of the legs and scrotum, the latter two being important symptoms. 2.

Distention of the veins of the abdominal and thoracic walls. 3. Jaundice and alcoholic stools. 4. Dysuria and diminution in the amount of urine.

*Gastric Symptoms.* Loss of appetite and vomiting are of frequent occurrence. The vomited matter may contain bile, especially if the tumor is high up, but vomiting of blood is exceptional, except when as a terminal symptom. In our fifth case hæmin crystals were found in the vomitus comparatively early in the disease. In Stern's congenital case meconium was present. The gastric pains have been referred to.

*Intestinal Symptoms.* There is generally some disturbance in the movements of the bowels, although the exact character varies. Thus there may be always constipation or always diarrhœa. There may be first constipation, and then diarrhœa, or they may alternate. More characteristic is an early diarrhœa followed by persistent constipation. The constipation is not absolute, except when some complication is present, as already referred to. Rarely "erections of the intestine" may occur (Schmidt). The movements may contain pus and blood, although the admixture of blood generally speaks for involvement of the large intestine.

*Hepatic Symptoms.* The liver has generally been described as not being enlarged, but in three of our cases there was quite marked increase in size. Rarely jaundice is present.

*Pulmonary Symptoms.* Dyspnœa is not uncommon, being due to plural effusions, the abdominal distention, or the weakness and anæmia. The plural effusion is bilateral or unilateral, and the fluid is serous or turbid or hemorrhagic.

*Urinary Symptoms.* The urine may be scanty and contain much urates. Albumin is frequently present, pus and blood rarely. There may be difficulty in urination of

even marked degree, as in our second case. The urination may be very frequent and painful.

*Cutaneous Symptoms.* Sweating is not uncommon, especially at night. The oedema of the legs and scrotum, due to pressure or hydræmia, may occur quite early. There may even be a slight anasarca. The veins of the abdominal wall and thoracic walls may be prominent. Of great importance is the appearance of a peculiar white color in the face, which may come on suddenly, and usually appears after the abdomen is distended. This rather characteristic color appeared in our first two cases very early, but in the fifth case it was noted a few days before death.

*External Lymph Nodes.* These are not generally enlarged. But in a case of melanosarcoma of the ileum, reported by Treves, there was a swelling in the left groin which was suspected of being a hernia, but which proved to be a lymph node containing metastases.

*Temperature.* The temperature may be quite normal throughout the disease, but is more common to find evening rises to  $101^{\circ}$ ,  $102^{\circ}$ , or  $103^{\circ}$ . Our cases all had febrile movements.

*Emaciation and marked loss of strength* are generally prominent features. They were present in all our cases, but developed in the fifth case very late.

*Blood Changes.* Here there is nothing characteristic, although anæmia and leucocytosis have been described. Schmidt made a careful examination of the blood in two cases, noted in both a diminution in the amount of fibrin and an increase in the number of hæmatoblasts. In our second case, an examination showed a moderate secondary anæmia, with slight polynuclear leucocytosis.

*Course of the Disease.* A consideration of the symptomatology as just given will show that the descriptions given by Baltzer and Nothnagel will not fit many of the cases.



I have therefore endeavored to classify the varieties of the disease in the following way:

1. Latent cases, the disease being first discovered at autopsy.
2. Cases with the clinical picture described by Baltzer, either the general symptoms, the distention of the abdomen, or the tumor being first noted.
3. Cases in which the first symptoms are due to an intussusception, or other variety of intestinal obstruction, or to perforation.
4. Cases resembling tubercular peritonitis.
5. Cases in which jaundice is the first symptom.
6. In one case there was the closest resemblance to an ovarian cyst.
7. Finally, the cases may bear a very close resemblance to appendicitis, an observation made for the first time in our cases.

DIAGNOSIS. Of all the symptoms just detailed the following are important for the diagnosis: the presence of a tumor (rectal examination) which is not markedly tender, abdominal distention, absence of symptoms of stenosis, early œdema of the legs, the lack of involvement of the external lymph nodes, the emaciation, the peculiar color, the absence of marked ascites. But none of these symptoms is absolutely constant. It is very difficult to establish any exact rules for diagnosis, as any one observer is apt to see but few cases. We shall endeavor to give a few hints and to indicate the diseases with which intestinal sarcoma is most likely to be confounded, omitting the very rare conditions such as actinomycosis or echinococcus disease.

The main question that arises is, Can these tumors ever be positively diagnosed? Undoubtedly in many cases it would have been impossible to make the correct diagnosis. We believe, however, that in a certain number of

instances the correct diagnosis can at least be strongly suspected. If there is a large movable tumor present, or, better, one large and several small tumors, or, if beside, growths can be felt by rectum, with but little or no ascites, and with early œdema of the legs, and the peculiar color described, the diagnosis of sarcoma of the mesentery or omentum is very probable. If these symptoms occur in a person under fifteen the diagnosis is still more probable. Having made a diagnosis of peritoneal sarcoma, and the tumor being movable, the existence of a primary intestinal sarcoma must be strongly suspected, and can be made positively if there exist early in the case intestinal disturbances, or if the colon bacillus can be isolated from effused fluid, if such be present (as in our fifth case). Of course, we can never say positively that the tumor is not primary in the mesentery. However, as far as treatment is concerned, there is no importance in this distinction.

The following are the diseases to be differentiated:

1. *Carcinoma of the Intestine and Peritoneum.* Baltzer states that sarcoma occurs earlier in life, that carcinoma is apt to produce a stenosis earlier, is more tender, and lasts longer. Schmidt believes that œdema of the legs, with little or no ascites, would favor sarcoma. This latter statement is certainly true. Baltzer's remarks are, however, not absolutely correct. We shall later see that, although sarcoma is usually rapid in its course, it may be slow, and there are numerous instances in which carcinoma runs a rapid course. Again, we have shown that sarcoma does occur after the fortieth year, although not commonly. According to our personal experience, carcinoma of the intestine occurs quite frequently in people between the ages of fifteen and twenty-five. It is true that it then generally involves the cæcum, descending colon, or rectum, but if metastases are already present

(and it is mainly under such conditions that the differential diagnosis must be considered) the cases might well be confused with sarcoma of the intestine. We will therefore modify Baltzer's statement, and say that under fifteen years of age the diagnosis would be decidedly in favor of sarcoma (although it is true that even congenital intestinal carcinoma has been described as well as congenital sarcoma), and that after the age of forty sarcoma is less probable but cannot be excluded. Important points against the diagnosis of carcinoma are the absence of external glandular involvement, the absence of tenderness, and the large size of the growths in the sarcoma cases.

2. *Tubercular Peritonitis, and Tuberculosis of the Mesenteric Lymph Nodes.* The differential diagnosis may be very difficult, and even if a positive diagnosis of sarcoma is made the presence of a concomitant tuberculosis cannot be excluded. This was made especially clear in a case described by Nothnagel, in which his diagnosis wavered between these two conditions, and in which the autopsy revealed lymphosarcomatous growths, springing from the edges of cicatrizing tubercular ulcers. The presence of a very large tumor or tumors speak more for sarcoma. The facies is different in the two conditions, but the recognition of this point requires much experience. Ascites is more apt to occur early in tuberculosis. A tubercular history is of no use in excluding sarcoma, nor is the existence of fever. If the ascetic fluid should reveal tubercle bacilli, of course the diagnosis of at least a tubercular condition being present would be absolute.

3. *Intestinal obstruction, intussusception or intestinal perforation when due to sarcoma,* is accompanied by the same symptoms as under other conditions, and the diagnosis can be made only if the growths can be felt and if other symptoms are present.

4. *Sarcoma of the kidney* is not generally so mobile.



Hæmaturia would speak decidedly for a renal growth, but this does not occur in the majority of cases. In renal sarcomata the tumor is generally located more on one side of the abdomen, but this may also occur in intestinal sarcoma. If nodules are felt elsewhere in the abdomen they speak for intestinal or mesenteric sarcoma, as the renal cases do not show metastases in the peritoneum. Further, the kidney sarcomata are less rapid in their course.

5. *Ovarian Tumors and Cysts.* A pedunculated intestinal growth may closely simulate an ovarian cyst, and, on the other hand, an ovarian tumor may be located in the upper part of the abdomen, and simulate a mesenteric or intestinal tumor. The finding of a pedicle (Hegar's method) springing from the uterus would make the diagnosis clear. In a case recently seen no pedicle was felt (Hegar's method not being tried), and as the tumor was surrounded by a group of distinct nodules, the diagnosis of intestinal or mesenteric sarcoma seemed assured. The operation, however, revealed an endothelioma of the ovary, and what had appeared to be separate nodules were found to be large irregularities springing from the tumor.

6. *Neoplasms of the Bladder and Prostrate Gland.* Given a large tumor in the region of the bladder or prostate, especially in a person under forty, it is necessary to determine whether or not the same is due to a secondary growth from an intestinal sarcoma or is a primary intestinal growth which has become adherent in the pelvis. In two of our cases some of the symptoms might easily have been construed as indicating a primary growth of the bladder.

7. *Retroperitoneal Sarcoma.* The differential diagnosis may again be very difficult here, although unimportant, for an intestinal tumor extensive enough to

resemble one of these retroperitoneal sarcomata is generally a non-operable case. We cannot enter into a description of these sarcomata here, but would refer to a review of the subject by Steele, in *The American Journal of the Medical Sciences*, March, 1900, page 322. He says that in retroperitoneal sarcoma the colon lies in front of the tumor, that obstruction of the intestine is apt to ensue, and that pain in the legs and in the lumbar regions is characteristic.

8. *Appendicitis with or without Peritonitis.* Our third and fourth cases show how closely this may be simulated by an intestinal sarcoma (and it is not difficult to appreciate how much greater this similarity might be in cases where the sarcoma is primary in the cæcum). The diagnosis will have to be made on the lines laid down until more cases are reported. I would lay great stress, however, on the attempt to find nodules by rectal examination. A single mass felt by the rectum is not of much use for differentiating the conditions, as such a finding is frequently enough made in appendicitis cases, and it not uncommonly occurs that a separate, very hard mass closely resembling a tumor may be felt by the rectum in cases of appendicitis.

9. *Differential Diagnosis between Lymphosarcoma and Other Varieties of Sarcoma.* This cannot at present be made with any degree of certainty; all we can now say is that with spindle-celled sarcomata there is apt to be one large mass, whereas the lymphosarcoma cases usually present multiple growths.

• EXPLORATORY LAPAROTOMY. We believe that this is indicated in all cases except in those in which there can be felt several distinct masses at some distance from each other. A large mass with nodules nearby may represent a solitary tumor only, as was demonstrated in the case of ovarian endothelioma cited above.

DURATION AND PROGNOSIS. Baltzer puts the duration at from two weeks to one and three-quarter years, most cases dying within nine months. This statement is corroborated by the cases since reported with the exception of a case described by Rutherford, which was of two and one-half years' duration. In our third case the history was only of one day's duration, but the statement made by the relatives that the patient had slight pains in the stomach for three months, although he continued at work, must be taken to indicate a longer existence of the illness.

The prognosis seems to be almost invariably fatal. A number of these cases have been operated on, and some, it is claimed, with a favorable result, but the ultimate outcome of the cases has not been given. In only one instance—a case reported by Babes and Nanu—was the patient alive after one year, and the authors themselves state that the ultimate outlook was bad. The other operated cases are as follows: Nicolaysen: spindle-celled sarcoma; recurrence in twenty-four days. Zuralski: cystic sarcoma; recovery for the time being. Lanne-longue: variety of tumor not given; ultimate result not stated. Heinze: resection of 110 cm. of the intestine with mesentery; recovery, but ultimate result not given. Among Baltzer's cases there are four in which death resulted within twenty-four hours after operation. Siegel has recently reported a case of lymphosarcoma for which he resected 30 cm. of small intestine, but death resulted within two weeks. Van Zwahlenburg (verbal communication: case to be presented at the meeting of the American Medical Association, June, 1900) has operated on a case, the patient being alive now, seven months since the time of operation.

TREATMENT. (a) *Operative*. When the growth can be removed completely this should certainly be done;



but we believe that cases of lymphosarcoma with extensive metastases should not even be subjected to exploratory incisions, as this is likely to hasten the occurrence of the fatal issue.

(b) *Medicinal.* In the literature there are a number of undoubted instances in which sarcomata, particularly lymphosarcomata, have been cured or improved by arsenic given internally, used hypodermatically, or parenchymatously (into lymph nodes). Such cases have been reported by Liebmman, von Ziemssen, Köbner, Billroth, Winiwarter, Tholen, Arning, and Wunderlich. In their cases there was generally present a multiple lymphosarcomatosis, or sarcoma of the skin. We believe that this treatment should invariably be tried in cases of intestinal sarcoma, and that it should be used also for the patients upon whom successful resections have been performed. Whether Coley's fluid would be of any value in these cases future experience alone can decide. The remainder of the treatment is purely symptomatic.

ADDENDA. Since writing the above my attention has been drawn to an interesting case reported by Lindner in the *Beiträge zur klin. Chirurgie*, 1899. This case was that of a man, aged thirty-six years, who six months before he came under observation was operated upon for a supposed carcinomatous ulcer of the head, and who for fourteen days was suffering from symptoms of ileus. There were present a marked enlargement of the lymph nodes on one side of the neck and a tumor in the right hypochondrium. At the operation an intussusception of the small intestine was found, and the gut was full of lymphosarcomatous tumors. A resection was done, but the patient died after twenty-four days, Lindner believes that the tumor was primary in the intestine, and that the ulcer on the head, which had been supposed to be carcinomatous, was probably sarcomatous. He

reports the case as an instance of a tumor whose metastases caused symptoms long before the primary growth was revealed, but does not state how long the tumors were present in the neck.

A case of intestinal sarcoma was reported to the Surgical Section of the New York Academy of Medicine by Dr. Weir, on May 14, 1900, for which he resected eight feet of small intestine. The patient lived for two days. Dr. Weir kindly allowed me to refer to this case before its publication in the Society reports.

Two further cases of lymphosarcoma occurred in the hospital in June, 1900.

#### LITERATURE.

##### (a) *Of Sarcoma of the Small Intestine.*

1. BALTZER: *Archiv. für klin. Chirurgie*, xiv., p. 717.
2. MADELUNG: *Centralblatt für Chirurgie*, 1892, No. 19, pp. 617-619.  
This is an abstract of Baltzer's conclusions.  
The next thirteen references are from Baltzer's article.
3. SOLOMON: *Charité-Annalen*, 1878, v. 140. Case not used by Baltzer.
4. HENOCHE: *Charité-Annalen*, x., p. 557. Case not used by Baltzer.
5. BECK: *Prager Zeitschrift für Heilkunde*, 1884, v., p. 442. Case not used by Baltzer.
6. WALLENBERG: *Berliner klin. Wochenschrift*, 1864, p. 497.
7. WALDENSTRÖM OU AKERBERG: *Uppsala lakareforen. Fördhan*, v., p. 3888.
8. MOXON: *Transactions of the Pathological Society*, London, 1873, xxiv., p. 101.
9. BESSEL-HAGEN: *Virchow's Archiv.*, xcix., p. 99.
10. PICK: *Prager med. Wochenschrift*, ix., p. 96.
11. BECK, HUGO: *Vide* 5, p. 447.
12. NICOLAYSEN CHRISTIANI HELBREDELSE: *Norsk. Magaz. for Lægevid.*, R. 3, vol. xv.
13. HAAS: *Wiener med. Presse*, 1886, xxvii., p. 471.
14. KRAUZ: *Prager med. Wochenschrift*, 1886, ix., p. 109.
15. LEHMANN, MAX: *Dissertation*, Würzburg, 1888.
16. ZURALSKI: *Dissertation*, Königsberg i. Pr., 1889.

The following cases I have collected: The first two were not used. In the former (17) there is no autopsy report; in the latter (18) there is no microscopical examination. They are included because Baltzer refers to them as possibly being of value. He could not obtain them.

17. EDWARDS: *Transactions of the Pathological Society of Philadelphia*, 1884, and *Philadelphia Medical Times*, 1882-1883.

18. HULBERT: *St. Louis Medical and Surgical Journal*, 1885, vol. xlviii.

19. MOLSON: *Canada Medical and Surgical Journal*, Montreal, 1881-1882.

20. LEGG: *St. Bartholomew's Hospital Reports*, vol. ii., p. 73.

21. PEPIN: *Journal de Med. de Bordeaux*, 1891, xxi., pp. 248-250.

22. ROLLESTON: *Transactions of the Pathological Society*, London, 1892, p. 67.

23. MOORE: *Transactions of the Pathological Society*, London, 1892-93, xxxiv., p. 99.

24. PERRY: *Transactions of the Pathological Society*, London, 1892-1893, xlv., p. 89 (secondary sarcoma of intestine).

25. JALLAND: *Lancet*, London, 1894, i., 1007.

26. GENERSIICH: *Pester med.-chir. Presse*, Budapest, 1893, xxix., pp. 390-393.

27. FLEMING and J. L. STEVENS: *Glasgow Medical Journal*, 1893, xxxix., pp. 455-457; also *Transactions of the Glasgow Pathological and Clinical Society*, 1891-1893, iv., 206-209.

28. RUTHERFORD: *Ibidem*.

29. FLEXNER: *Johns Hopkins Hospital Reports*, 1893, p. 153.

30. STERN, CARL: *Berliner klin. Wochenschrift*, xxxi., No. 35.

31. ROVSING: *Hospital-Tidende*, 1897, No. 46.

32. HEINZE: Dissertation, Greifswald, 1897. Zur Casuistik des primären Dünndarmsarkoms.

33. HAMMER: *Prager med. Wochenschrift*, 1896, xxi., pp. 212-215.

34. SMOLER: *Prager med. Wochenschrift*, xxiii., pp. 135 and 139.

35. LANNELONGUE: *Journal de Med. de Bordeaux*, 1897, xxvii.

36. BABES and NANU: *Berliner klin. Wochenschrift*, 1897, xxxiv., 7.

37. SCHMIDT: *Wiener klin. Wochenschrift*, 1898, p. 505.

38. ABLON: Les fibromes embryonnaires (sarcomes) de l'intestine chez l'enfant, Paris, 1899.

39. DEBRÜNNER: Cited by Ablon.

40. BOAS: *Darmkrankheiten*, 1899, p. 352. (No cases belonging to author.)

41. NOTHNAGEL: *Erkrankungen des Darms u. Peritoneums*, 1898.

42. SIEGEL: *Berliner klin. Wochenschrift*, 1899, p. 770.

43. TREVES: *Intestinal Obstructions*, 1899, p. 268.

44. RÜSSAU: *Sarcom des Dunndarms*, Kiel, 1891, L. Handorff.

45. ORTH: *Ueber die Sarcome des Darms, Mesenteriums u. retroper. Raums*, Tübingen, 1890.



The following articles could not be utilized:

46. ENGSTRÖM: *Fenska läkarsällsk. handl.* Helsingfors, 1897, xxxix., 806-925.
47. PIOTROFF (n. v.): Tumors (lymphosarcomata and myomata of small intestine), *Lactoprussk. chir.*, St. Petersburg, iii., 110-113.
48. CHERNYAKHOOSKI, M. G.: *Khirurgia Mosk.*, 1898, iii., pp. 583-601.
49. WESTERMARK: *Nordisk. med. Arkiv.*, 1899, x., No. 26.
50. FERRARESI: Linfoma diffuso primitivo dell. Intestino d. Osp. di Roma, 1891-1892, xi., 185.
51. STEINER: Heidelberg Pathological Institute, 1885.
52. QUENSEL, ULRİK: *Nordisk. med. Arkiv.*, 1898, No. 30.

### *Discussion.*

Dr. HARLOW BROOKS said that he had seen only one case of this kind, occurring in a stoker on an ocean steamer. He had felt slightly ill on reaching port, and had gone to St. Vincent's Hospital, where he died three days later. The autopsy had shown a very extensive involvement of the small intestine, and some involvement of the liver.

### NOTES ON ACID INTOXICATIONS.

Drs. C. A. HERTER and A. J. WAKEMAN presented a paper on this subject, which was read by Dr. Herter.

Organic acids appear in the urine in pathological quantities in a number of derangements; in diabetes, in some acute and chronic disorders of digestion, in arthritis deformans, in acute yellow atrophy of the liver, in poisoning by phosphorus and perhaps in scurvy. When such acids are present in the urine in excessive amount it is owing either to the increased formation of organic acids in the digestive tract, or in the tissues, or to an impaired capacity of the organism to burn acids produced in digestion or metabolism in not excessive quantity. Or it may happen that both these factors—increased production and diminished combustion—are combined.

Our knowledge of the pathological conditions under which the urine comes to contain organic acids in excess is still in the first stages of development, but we know enough to show us that the processes which have this effect are well worthy of study. At the present time it is an established fact that in all severe cases of diabetes the blood and urine contain certain organic acids, and especially  $\beta$ -oxybutyric acid, in excess. This fact is not merely of academic interest, but has a deep, practical, clinical interest; for it is true that a large accumulation of organic acid in the blood leads to the robbing of the organism of its alkali and that ultimately coma sets in as a consequence of the diminished alkaliescence of the blood which follows the withdrawal of sodium and potassium from the blood. We may appropriately speak of a patient in diabetic coma as suffering from an extreme degree of acid intoxication.

But our interest as clinicians in states of acid intoxication is by no means limited to the state of diabetic coma. In the published observations made by one of us (Herter), on a variety of cases of diabetes it is clearly shown that organic acids may appear in the urine a long time before the development of coma, and that the prognosis in a particular case of diabetes can be more accurately prophesied from a knowledge of the excretion of the organic acids than from the percentage or quantity of sugar in the urine. This fact appears to us in itself of sufficient practical importance to make it desirable to bring our method of balancing the acids and bases of the urine to the notice of clinicians and workers in clinical laboratories. As regards the practical utility of this method in the study of other diseases than diabetes, we have not had sufficient experience to form a judgment. It may, however, be said that the method is applicable whenever the amount of organic acid excreted in a day is considerable, say not less

than would suffice to neutralize one gram of sodium. Where the amount excreted is smaller, other methods of investigation are to be preferred.

When we speak of balancing the acids and bases of the urine, we refer to determining the basic values of the chief known bases of the urine and comparing them with the corresponding basic values of the chief known acids of the urine. In a state of health the known bases of the urine suffice very nearly to *balance* the known acids. In a state of acid intoxication, with the excretion of organic acids in considerable amount, there is failure of the known acids to balance the known bases; the bases are in excess of the amount required to neutralize the acid, and from the amount of their excess, *which we know*, we can calculate the amount of the organic acid or acids in the urine. This fact forms the basis of our method.

An example will make our meaning clearer. The following values were obtained for the acids and bases in the twenty-four hours' urine of a robust woman, aged sixty, and will serve as a type of the normal.

ACIDS	(IN TERMS OF SODIUM)	BASES
SO <sub>3</sub> (preformed) =	.9734 gms.	K <sub>2</sub> O = .9232 gms.
SO <sub>3</sub> (combined) =	.0356 "	Na <sub>2</sub> O = 3.8810 "
P <sub>2</sub> O <sub>5</sub> (dibasic) =	.7432 "	CaO = .2154 "
P <sub>2</sub> O <sub>5</sub> (monobasic) =	.1987 "	MgO = .0806 "
Uric Acid =	.0584 "	NH <sub>3</sub> = .4704 "
Cl =	4.4760 "	5.5706 "
	6.4853 "	

It will be observed that here we have taken into account sulphuric acid (SO<sub>3</sub>, preformed and combined), phosphoric acid (P<sub>2</sub>O<sub>5</sub>, monobasic and dibasic), hydrochloric acid (Cl) and uric acid. Opposed to these acids are the oxides of the bases sodium (Na<sub>2</sub>O), potassium (K<sub>2</sub>O), calcium (CaO), magnesium (MgO), and ammonia (NH<sub>3</sub>). It will also be noticed that the total amount of acid



(6.4853 gms.) more than suffices to neutralize the total amount of the bases (5.5706 gms.). As normal urine contains neither free acid nor free base, it is proper to assume that there exist in the urine one or more unknown bases (*i. e.*, neither Na, K, Ca, Mg or  $\text{NH}_3$ ) which neutralize the apparent excess of the known acids.

Let us now contrast the results from normal urine, with the results obtained in a case on the verge of diabetic coma. In this particular case the twenty-four hours' urine gave the following values.

ACIDS	(IN TERMS OF SODIUM)	BASES
$\text{SO}_3$ (pref.)	= 1.3350 gms.	$\text{K}_2\text{O}$ = 3.2610 gms.
$\text{SO}_3$ (comb.)	= .0557 "	$\text{Na}_2\text{O}$ = 3.5780 "
$\text{P}_2\text{O}_5$ (dib.)	= 1.3610 "	$\text{MgO}$ = .1231 "
$\text{P}_2\text{O}_5$ (monob.)	= .3402 "	$\text{CaO}$ = .2300 "
Uric Acid	= .1469 "	$\text{NH}_3$ = 6.6400 "
Cl	= 3.5780 "	13.8321
	6.8168	

Here it is clear that the total bases of the urine are markedly in excess of the total acids. The excess of bases over acids is equivalent to 7.0153 gms. Na. But since the excess is only apparent, owing to the fact that the urine contains no free bases, the excess of base must be united to some acid or acids in the urine, *not* to be one of the known inorganic acids. The acid can, therefore, only be an organic acid. If we suppose this acid to consist wholly of  $\beta$ -oxybutyric acid (which is not strictly true) we may say that the organic acid of the urine is equivalent to 31.7 gms. of  $\beta$ -oxybutyric acid, since 1 gm. Na corresponds to 4.52 gm. of this organic acid.

It is well known that when an acid in considerable amount enters the blood from the cells of the body or from the digestive tract it is promptly neutralized by ammonia, derived either from the normal decompositions of the cell proteids or from proteids of the food. This

ammonia is under normal circumstances destined to enter into the formation of urea, but is diverted from this fate by union with the acid, to which reference has just been made. The ammonium salt of the acid resulting from this union passes from the blood into the urine and thus rids the organism of the excess of acid. This process constitutes a highly important chemical defense against intoxication of the organism from acids, a defense much more effective in carnivorous and omnivorous animals than in herbivora, which can supply only a limited amount of ammonia.

The diversion of ammonia for the neutralization of acids saves to the organism the bases, sodium and potassium, most essential in preserving the normal alkalescence of the blood, a condition essential to the maintenance of life. Only after the resources of the body in ammonia have been exhausted by a persistent and increasing quantity of acid is the blood robbed of its sodium and potassium, and the cells of their calcium and magnesium.

Hence it is that in disease attended by the elimination of excessive amounts of organic acids it is chiefly the ammonia of the urine which we find increased among the bases present there. The fact that this enables us to judge roughly of the amount of acid that is leaving the body day by day, and many observers judge of the severity of an acid intoxication by the amount of ammonia in the urine. When it is not practicable to carry out the balancing of the acids and bases in the manner described in this paper, a knowledge of the nitrogen of ammonia in the urine is often helpful, but it is a much less reliable guide to the amount of acid excreted than the more elaborate method we have described. This is because the ammonia of the urine varies so widely in health, mainly owing to differences in diet. In health the urine contains from 1-6 per cent. of its nitrogen in the form of nitrogen

of ammonia. But since a considerable amount of organic acid may be leaving the body without causing the presence of more than 6 per cent. of nitrogen of ammonia, it is easy to see how the presence of the acid would be overlooked if we relied on the determination of the ammonia alone. Similarly, if the urine contains 8 per cent. of N of  $\text{NH}_3$  most of this may be united to the normal acids of the urine or most of it may be united to a pathological organic acid. The truth can be learned by a balance of the acids and bases. Still it is only fair to say that when the N of  $\text{NH}_3$  exceeds 10 per cent. persistently the body is eliminating large amounts of organic acid. When the percentage rises to 20 the patient is usually out of danger of coma.

In any determination of the organic acids by the method we have described it is essential to employ urine in which the urea has not undergone any decomposition. If therefore the urine cannot be obtained quite fresh, it should be carefully preserved by the addition of phenol or preferably chloroform, in which case the urine should be occasionally shaken. Only acid urines should be used except those which may be neutral when voided.

It is often convenient to express the excess of bases (indicating the amount of the unknown acid) in terms of the organic acid. In the case of diabetes, the chief acid being  $\beta$ -oxybutyric, we can make use of the factor 4.52 to give us the amount of acid in terms of this particular acid.

The idea of balancing the acids and bases for the purpose of recognizing the presence of organic acids in the urine appears to have originated with Stadelman.<sup>1</sup> Magnus-Levy<sup>2</sup> has recently made use of the principle for the

<sup>1</sup> Stadelman, "Ueber den Einfluss der Alkalien, U. S. W.," Stuttgart, 1890.

<sup>2</sup> Magnus-Levy, "Die oxybuttersäure" und ihre Beziehungen zum Coma diabeticum. *Arch. f. exper. Path. und Pharmacol.*, Leipzig, 1899, Bd. 43, S. 149.



purpose of studying the urines of patients suffering from diabetic coma, but the method employed by him and by Stadelman is much longer and more laborious than the one which we have described and cannot be said to possess any distinct advantage for clinical use except where very small amounts of organic acid are being excreted.

---

*Stated Meeting, May 9, 1900.*

EUGENE HODENPYL, M.D., PRESIDENT.

A CASE OF TUBULAR CAST OF THE BRONCHUS.

Dr. THEODORE C. JANEWAY presented this specimen. It had been coughed up by a middle-aged and very stout gentleman, who had been sick for about ten days with what had been supposed to be the grippe. He had been apparently convalescent, when he had been suddenly seized with dyspnœa and increased fever. After some hours he had expectorated this cast, which was perfectly tubular and of about the size of the main bronchus. At its lower end it showed the beginning bifurcation into the smaller bronchi. The specimen not having been seen until it had been immersed for some time in alcohol, no cultures had been made. The man died a few days later, having exhibited signs indicative only of bronchitis with marked dyspnœa and cyanosis. On section, the cast showed a fibrinous exudate with numerous cell nuclei. Scattered through it were clumps of round-ended bacilli, which in their morphology and staining exactly resembled diphtheria bacilli. The speaker said that two cases had been reported in the London Pathological Society. One of these had been reported by Dr. Pye Smith in 1880. In this instance a woman had expectorated a cast of the trachea

and primary bronchi. At the autopsy the trachea and bronchi were found lined with tubular casts. Rod-like bacteria were present. The other case had been reported by Dr. Murchison. A prominent laryngologist had seen the case just reported, so it seemed safe to assume that no evidence of diphtheria had been visible during life, though it was apparently a case of diphtheria of the bronchi.

#### DISSECTING ANEURISM OF THE AORTA.

Dr. H. S. CARTER presented this specimen, which had been taken from a man, aged eighty years, who had died some months ago in Bellevue Hospital from chronic interstitial nephritis. At the autopsy the condition in the aorta had been discovered accidentally, and for this reason the specimen was not in perfect condition. The dilatation of the aorta started from the sinus of Valsalva. The dissecting aneurism began at the junction of the descending part of the arch and thoracic aorta and continued through into both common iliacs. The left suprarenal was given off directly from the aneurismal sac, as was also the inferior mesenteric artery. Dr. Carter said that up to 1856 only eight cases had been reported; up to 1895 there had been seventeen, and by 1897 two hundred cases had been collected, including all dissecting aneurisms, but the greater part of these had been of the arch of the aorta. There was only one case on record in which the diagnosis had been made during life, and this had been confirmed by autopsy. It should be noted that the history of these cases had been pretty uniform—a history of sudden pain in the chest and back and shooting into the abdomen, but not radiating down the arm as in the angina pectoris. There was also a feeling of something having given away. If the patient did not die immediately, there were œdema and other evidences of disturbed circulation.

*Discussion.*

Dr. JAMES EWING said that he had studied the specimen a good deal. At first it had appeared to be a case of true double aorta, largely because of the large vessels given off unquestionably from the aneurism. Though not familiar with the literature it seemed to him rather difficult to explain the separation of a trunk of the size of the inferior mesenteric from the aorta, and the subsequent canalization that had undoubtedly taken place in this case, if it was one of dissecting aneurism. A strong point in favor of dissecting aneurism was the presence of three openings at the top of the sac. He asked if Dr. Carter had seen in his reading any description of the manner in which these large vessels were given off.

Dr. CARTER replied that in looking over the literature of the subject he had found a number of similar specimens reported, particularly descriptions of the interior mesenteric given off from the aneurismal sac.

A CASE OF NON-TRAUMATIC RUPTURE OF THE HEART.

Dr. N. B. POTTER presented this specimen, which had been removed from a large, stout woman, admitted to the City Hospital on February 28th. According to the history she had suffered for five or six years from bronchitis and asthma. Some time in last November bronchitis had become more than usually troublesome, and she had begun to complain of pain in the left side and back, radiating to the shoulder and down the arm. When first seen by him on March 1st, she had been pale and cyanosed, and there had been much cough and expectoration. The physical examination had revealed nothing further than diffuse bronchitis. The heart sounds were rather feeble. In spite of stimulation she became gradually more cyanosed, and finally died, about two weeks after admission.



At the autopsy the pericardium had been found fairly full of blood, much of which was old and clotted. It was estimated that this represented about twenty ounces. A rupture was found in the left ventricle, a short distance from the interventricular septum. On the ventricular side was a large thrombus attached at the superior extremity. It formed a sort of valve and occluded the opening. Apparently the rupture had taken place some days before death. The wall of the ventricle was remarkably thin.

### *Discussion.*

Dr. G. LANGMANN thought it was not at all uncommon for persons to live for days, or even weeks, after rupture of the heart. He referred to the case of a shoemaker, who, while moving some furniture from one room to another, had been seized with distressing pain over the heart necessitating his going to bed. This pain had gradually subsided, and he had resumed his work. About two weeks later there had been a second severe attack of pain, and physical examination at that time had shown only slight increase in the cardiac dulness, and a feeble and small pulse. The patient had died about four hours later, and the autopsy had revealed not only the fresh rupture in the left ventricle, but an older rupture in the right ventricle. From its appearance and from the history this rupture had evidently occurred two weeks previously. Both ruptures were about midway between the apex and the base of the heart. The walls were thin, but there was no marked fatty degeneration.

### THREE CASES OF TUBERCULOUS PERITONITIS.

Dr. LEON T. LEWALD reported these cases and presented specimens. The first case was that of a man, aged

thirty-five years, who had presented at autopsy a tuberculosis of the seminal vesicles and of the epididymis on the left side. The tuberculous process had apparently started in the left epididymis and spread to the prostate. On opening the peritoneal cavity a small quantity of dark fluid had been found, and the peritoneum had been generally studded with tubercles of large size. Miliary tubercles were scattered over the peritoneum covering the intestine, and they were particularly abundant on the under surface of the diaphragm. There was also a rather recent miliary tuberculosis of the lungs. The oldest-looking lesions were in the region of the prostate and the seminal vesicle, though it was not at all unlikely that some small focus in the lung might have been the original process, but the peritoneal tuberculosis was secondary to the genital tuberculosis. The second case was that of a man, aged fifty years, who had been treated for cirrhosis of the liver in the Roosevelt Hospital last December. He had been operated on with the idea of determining whether he had a tuberculous peritonitis or an accumulation of fluid entirely the result of the disease of the liver. The operation had revealed nothing more than the cirrhosis of the liver. On March 31st, the man's abdomen had been tapped, and fluid removed, and this had failed to show any tubercle bacilli present. On April 4th he had died, and the autopsy had shown a well-marked tuberculous peritonitis. The lungs showed a chronic tuberculosis. The third case was that of a man, aged thirty-five years, who had been suffering from alcoholism and cirrhosis of the liver. The autopsy showed a miliary tuberculosis of the peritoneum. A careful search had failed to show any other tuberculous process in the body, so that the case was considered to be one of primary tuberculosis of the peritoneum, associated with a marked cirrhosis of the liver. While primary tuberculosis of the peritoneum was quite rare, it was

somewhat less so when associated with cirrhosis of the liver, as shown by statistics published by Osler. Ziegler mentioned the occurrence of primary peritoneal tuberculosis, although the manner of its occurrence was not understood.

#### A CASE OF ADDISON'S DISEASE.

Dr. LEWALD also presented specimens from a case of Addison's disease occurring in a woman fifty years of age. The case was typical, so far as the bronzing of the skin was concerned. The skin of the face and neck was of a very dark bronze, and the skin on the hands, wrist, and forearms of a somewhat lighter tint. The body was emaciated. The bronchial glands were markedly enlarged and pigmented. The heart was small and showed brown atrophy. The coronary arteries showed very slight arterio-sclerosis. There were old adhesions about the stomach, liver, and spleen. In the large intestine were some cicatricial areas in the mucous membrane, apparently the result of some old ulcers. There were also a few lymphatic nodes, which were apparently tuberculous. The right suprarenal was of about twice the normal size, and was adherent to the under surface of the liver. On section, it had been found to be almost entirely replaced by caseous masses. The left suprarenal was surrounded by a mass of adhesions, which bound it to the pancreas. A small abscess had formed between the pancreas and the suprarenal gland. Its contents were cheesy, and the suprarenal gland itself was largely replaced by cheesy nodules. Sections were exhibited under microscope.

#### *Discussion.*

Dr. CARLIN PHILLIPS asked, in the case of supposed primary tuberculous peritonitis, whether the small nodules



contained tubercle bacilli or not. About four or five years ago Wagner described a form of fibroid peritonitis occurring in cases of cirrhosis of the liver which at times resemble in the gross appearance miliary tuberculosis. This condition was termed pseudo-tuberculosis peritonitis.

Dr. LEWALD replied that one of the sections exhibited under the microscope showed typical tubercles, and he also had a specimen showing stained tubercle bacilli.

A CASE OF PAPILLOMA OF THE OVARY WITH SECONDARY  
DEPOSITS IN THE PERITONEUM.

Dr. G. LANGMANN said that the President had exhibited at the last meeting one feature of the interesting specimen that he was about to present. The specimen had been taken from a woman, seventy-three years old. He had known her for several years, and knew that she had a large tumor on the right side, apparently ovarian. Last October, he had been called to see her because of a considerable enlargement of the abdomen. Inquiry showed that she had had a severe fall last summer, and that since that time her health had failed, and the abdomen had been increasing in size. When first seen the size of the abdomen had precluded thorough examination of the abdominal organs. There was a very large, smooth tumor in the abdomen having a hard, blunt border running from side to side. On November 8th, the abdomen was tapped, and about five litres of clear, amber-colored fluid evacuated. Examination after the tapping showed one tumor about the size of a man's head on the right, and another about the size of a fist on the left side. Percussion showed that regular vesicular resonance ran into the tympanitic resonance of the colon. No trace of the liver could be found by percussion. A second tapping had been required in a short time, and bloody fluid had been removed

at this operation. Shortly after the third tapping she died. The autopsy revealed an immense ovarian tumor on the right side, and a smaller one on the left side. The liver had not been visible on opening the abdomen. All of the ileum and the omentum were matted together. The vesical and parietal peritoneum were covered with small nodules, as was also part of the surface of the ovarian cyst. The ovarian tumor was a papillary cystadenoma, and some of the cysts were already suppurating. Microscopical examination of the nodules by Dr. Hodenpyl had revealed nothing but ordinary papilloma. When these cysts broke and scattered their contents over the peritoneum nodules developed from direct implantation, so that the result was practically like that of a malignant growth. The fall that this woman had experienced had probably caused a rupture of some of the cysts, and in this way he explained the decline in her health. The autopsy also showed numerous stenoses in the colon, probably as a result of long-continued pressure. They were interesting because of the absence of symptoms pointing to such a condition. The numerous diverticula along the colon had been exhibited by Dr. Hodenpyl at the last meeting of the Society.

#### *Discussion.*

Dr. PHILLIPS stated that in the summer of 1896 Zeigler, of Freiburg, presented to his advanced class in pathology a similar tumor which histologically was a papilliferous cystadenoma of the ovary. He considered the tumor of very great importance, as it was histologically benign but clinically malignant and capable of metastasis.

Dr. HODENPYL objected to looking upon the case just reported as a malignant growth. It was primarily a connective tumor, and therefore he could classify it as a benign tumor.

Dr. LANGMANN replied that one might make a distinction between clinical malignancy and microscopical malignancy. He thought no one would be justified in recommending the early removal of such a cyst because of the possibility of just such an accident as had occurred in this case.

A DEMONSTRATION OF NUCLEIC ACID EXTRACTED FROM  
BACILLUS TUBERCULOSIS.

Dr. P. A. LÉVENE reported results of studies in this direction. The products exhibited had been procured from cultures made on synthetic media, *i. e.*, such as were composed of mineral salts and were free from proteid material. The speaker said that all of the nuclear compounds were composed of nucleic acid. Until recently it had only been known that they contained phosphoric acid, and that some of them contained xanthin bases. Only very recently had different nucleic compounds been recognized. It was natural to expect that cells producing diseases would probably store up its power of producing disease in the nuclei, and probably in the nucleic acid. Nucleic acid was more active than the nuclear compound itself. Last year he had presented a paper on the chemistry of the tubercle bacillus, and it was only since then that he had succeeded in devising a method which would enable him to obtain nucleic acid from any cell or tissue whatever. In the tubercle bacillus one found a free nucleic acid, and the nucleic acid in combination with proteid material. He exhibited two samples, one a copper salt obtained from the free acid, and the other procured from the combined nuclein. The acidity in the growth was due partly to the nucleic acid. This could be demonstrated by the fact that nucleic acid had the property of precipitating any proteid, as, for instance, peptone. The precipitation with peptone was demonstrated. It had occurred to him that this nucleic acid



might be the cause of certain necrotic processes taking place around the bacilli.

ON THE PROPERTY OF PRECIPITATING ALBUMIN EXHIBITED  
BY SOME PATHOGENIC BACTERIA.

Dr. E. LIBMAN said that he had been surprised to find that nearly all pathogenic bacteria he had tested could precipitate serum-albumin and egg-albumin in the presence of sugar. The result depended largely upon the amount of acid formed. This reaction occurred if only 0.1 per cent. of glucose was present, and this percentage was present in the blood normally. Pneumococci did not precipitate albumin at all. These facts might possibly be of interest in explaining some lesions, especially in diabetics. This study opened up interesting possibilities in connection with acid toxæmiæ in the human body. Dr. Bookman had examined these tubes for him, and had reported that there was no doubt about the precipitate being albumin.

*Discussion.*

Dr. P. A. LEVENE thought the reaction was due to the presence of free nucleic acid.

Dr. LIBMAN said that if the acid was nucleic acid, he thought it should act even though sugar was not present.

ON THE INFLUENCE ON NORMAL ANIMALS OF BLOOD FROM  
ANIMALS DEPRIVED OF THEIR ADRENALS.

Dr. I. LEVIN made some remarks on this subject, and exhibited blood-pressure tracings. These tracings had been made to illustrate the influence on the blood pressure of injecting into a normal animal the defibrinated blood from an animal deprived of its adrenals five hours previously. The tracings showed a marked rise of blood

pressure lasting for a considerable time. As a control, tracings were shown of the blood pressure of an animal who had received an injection of defibrinated blood from a normal animal. It showed that there was scarcely any perceptible rise of blood pressure following this injection.

These tracings showed that the first injections resulted in a toxic condition. He thought the absence of the tonic action of the adrenals on the muscular and circulatory system was not sufficient to explain that fatal result of the extirpation of these glands.

### *Discussion.*

Dr. LANGMANN said that some experiments had been made with the suprarenal extract in connection with snake poison, and these animals had almost no adrenal tissue. Suprarenal extract obtained from sheep or oxen was injected into the guinea-pig poisoned with snake poison. These animals showed a much greater resistance to the snake poison. Experiment had shown that only the suprarenals possessed this power. It might be that the effect of the suprarenal extract on the heart and blood pressure was in itself sufficient to account for this increased resistance in the animal without supposing that the product of the adrenals was an antitoxin.

Dr. P. A. LEVENE said that from the suprarenal a substance had been obtained which produced a tremendous rise of blood pressure. If one accepted the theory of internal secretions, then by removing the gland the substance causing this increase of blood pressure was removed, and the blood of the animal should not contain the substance which caused the rise of the blood pressure. This was very different from what had been just described in the experiment of Dr. I. Levin.

*Stated Meeting, October 10, 1900.*

EUGENE HODENPYL, M.D., PRESIDENT.

SARCOMATOSIS OF THE PERITONEUM.

Dr. F. C. WOOD presented several specimens. The first of these was one of sarcomatosis of the peritoneum, removed from a small Chinese woman, who was very much emaciated. An exploratory operation had been done, and she had died soon afterward. The autopsy revealed the mass presented. The determination of the primary growth was especially difficult in this case because nearly all of the abdominal organs were involved. The spleen had escaped. There were no metastases anywhere in the body. Presumably the growth had originated from the retroperitoneal lymph nodes, but microscopical examination of the tumor was rather against this, for the appearance presented was that of a sarcoma consisting of round and spindle cells. The specimen under the microscope showed an extreme degree of fatty degeneration. The right ovary could not be found, so that possibly the growth might have started from that ovary. The uterus was not invaded. The bladder wall had been invaded, and was about one inch in thickness. The woman claimed to have been in perfect health up to five months before admission to the hospital, and stated that there had been no evidence of the tumor previously.

Dr. Wood then presented a case of

DIFFUSE LYMPHO-SARCOMA OF BOTH KIDNEYS.

The pancreas, liver, uterus, and ovaries were involved in this case. The specimens had been removed from a woman, twenty-six years of age, who had always been an



invalid. About one year and an half before admission to the hospital she had begun to have considerable pain in the abdomen, and about the same time had noticed some enlargement of the abdomen. The urine had been examined repeatedly for a long time by a competent physician, but with negative result, although it was assumed that the kidney was the seat of a new growth. On admission, there was an oval tumor in each flank. The urinary examinations in the hospital had also been negative. The blood examination showed moderate leucocytosis. The woman had died from shock the day after an exploratory laparotomy. The kidneys presented suggested foetal lobulation. When fresh, the tubular markings had shown pretty well, and portions of the kidney tissue seemed good on gross examination. Microscopical examination, however, showed that the kidney tubules were separated from one another by a layer of small round cells. One of the kidneys presented weighed one pound and nine ounces, and the other, one pound and four ounces. In this case the pancreas was large and quite firm, and microscopical sections showed infiltration of lymphoid cells into the trabeculæ. The ovaries were completely infiltrated, as was also the uterus. A large ragged ulcer, about three inches long, was situated in the ileum just above the caput coli, and extended around the entire circumference of the gut. There was nothing in the history pointing to such a condition. Sections of the ulcer showed nothing but necrotic tissue extending down in portions to the peritoneum. No tubercle bacilli were found, so that the cause of the ulcer was still in doubt. The spleen was perfectly normal, both in size and structure. The question naturally arose, Did not this patient have a simple pseudo-leukæmia? He believed one case of lymphatic leukæmia without enlargement of the spleen had been reported, yet the general

findings had not been those usually present in lymphatic leukæmia. He would, therefore, classify the infiltration in the case just presented as one of diffuse lymphosarcoma. Sections of the kidney were exhibited under the microscope.

The following specimens were then shown by Dr. Wood:

#### ACCESSORY LOBE OF THE LIVER.

There had been a congenital atrophy of a portion of liver tissue, thereby cutting off a segment. The patient had entered the hospital with pain in the abdomen and a high temperature. Physical examination of the chest had revealed a pneumonia. As there were jaundice and abdominal pain, cholelithiasis had been diagnosed. Palpation showed a mass thought to be the gall bladder, but this proved at autopsy to be an accessory lobe of the liver. Death had resulted from pneumonia.

#### CIRRHOSIS OF THE LIVER.

In this case, there were several adhesions between the anterior abdominal wall and the omentum, containing several large veins. The diaphragm was likewise adherent to the liver. The veins running from the liver to the umbilicus were also enlarged. The man was about fifty-five years of age, and up to three weeks before admission had been able to work. Nevertheless, the autopsy had shown advanced cirrhosis and advanced change in the portal circulation. The explanation probably was to be found in the existence of a complete collateral circulation. The operative cases in which a collateral circulation had been established had usually been relieved for a number of years. This operation consisted in stitching the omentum to the abdominal

wall, and thereby short-circuiting the portal circulation into the general circulation, and reducing the pressure in the portal system.

#### METASTATIC EPITHELIOMA IN THE HEART.

Regarding this case the speaker said that the peculiar feature was that there were no other metastases in any part of the body. The patient was a man who had previously had the tongue removed for epithelioma. The tumor had recurred in the supraclavicular lymphatic nodes, and death had finally resulted from pressure on the trachea. The autopsy had revealed infiltration of the tissues of the neck and involvement of the lymph nodes in the anterior mediastinum. Examination of the heart showed metastases deep in the muscle, and also superficially in the fat on the line of the vessels. They were probably embolic in origin. This condition of the heart had given rise to no symptoms.

#### *Discussion.*

Dr. L. A. CONNER said that he had been deeply interested in the diffuse sarcoma of both kidneys. He had not realized before that it was possible for such a growth of sarcoma to involve the kidneys, and still the markings to be preserved so well. This fact led him to suspect that there might be some other explanation.

Dr. WOOD replied that the picture under the microscope showed plainly that the cells had infiltrated the spaces between the tubules, and left a fairly good tissue between. The same thing was seen sometimes in cases of nephritis and also in the involvement of other organs. He did not assume that the tumor was primary in the kidneys.

Dr. E. LIBMAN said that last spring he had presented



some cases of sarcoma of the intestine. In one of these, the kidneys had been infiltrated in exactly the same manner, as shown by both gross and microscopical examination.

A CASE OF PULMONARY STENOSIS DUE TO ULCERATIVE  
ENDOCARDITIS OF AORTIC VALVE.

Dr. E. LIBMAN presented this case. The patient was a man, twenty-one years of age, who had been well up to May, 1897, when he had suddenly lost the sight of the right eye. He had recovered, some weeks later, sufficiently to be able to read large letters. Three months afterward he had lost the power of speech for twelve hours. On October 26, 1897, while sitting in a chair he had become paralyzed on the left side and totally unconscious. On admission to the Mount Sinai Hospital the following day he was found to be hemiplegic and unconscious. The left ventricle was distinctly hypertrophied. There was a rough, blowing systolic murmur at the apex, transmitted slightly to the left. There was a very loud systolic murmur over the aortic valve, which was transmitted upward and downward, and accompanied by a very marked thrill in both the carotids and subclavian veins. There was no elevation of temperature at this time, but about two weeks later he developed a fever, and this continued up to the time of his death. Six days before death, a blood culture (blood from vein) showed the presence of the staphylococcus albus. Two days before death, a systolic murmur and thrill developed in the pulmonary region, and were entirely distinct from those in the aortic region. The second pulmonic sound did not diminish in intensity. The clinical diagnosis was ulcerative endocarditis of the aortic and pulmonary valves successively. Petechiæ developed over the body. A culture from the heart's blood made directly post-

mortem after burning the skin also showed the staphylococcus albus. The post-mortem showed the right ventricle very markedly hypertrophied. The aortic valve was ulcerated, and in the free edge of two of the flaps and part of the third there were large vegetations. The sinuses of Valsalva were filled with vegetations. In the interventricular septum back of the aortic valve there was an enormous hemorrhagic swelling, which almost entirely occluded the pulmonary artery. Cultures from the vegetations and the centre of the swelling showed the staphylococcus albus. Spreads from the soft, purulent centre of the swelling showed numerous staphylococci.

*Discussion.*

Dr. G. LANGMANN said that all these or similar cases were mostly classified under the name of aneurism of the heart wall or septum, although they surely did not form a true aneurism. Their origin was justly attributed to an affection of the heart wall continuously hit by the infected valve.

Dr. LIBMAN replied that the stenosis had been due to the aortic endocarditis, as the latter had caused an enormous inflammatory swelling in the septum.

Dr. JAMES EWING asked what significance was attached to the presence in these cultures of the staphylococcus albus. It seemed to him this might have been an accidental germ in the circulation, or the specific cause of the endocardial lesion, or possibly a contamination of the culture. It was also possibly the result of an ante-mortem invasion of the blood by these germs.

Dr. LIBMAN said that there was an old endocarditis. Out of one hundred and fifty blood cultures he had never met with the staphylococcus albus as a contamination. From the technique employed, both during life and at

the autopsy, he felt positive that the staphylococcus albus was the specific cause. Furthermore, he had found bacteria in the blood (*intra vitam*) in fatal cases of ulcerative endocarditis, whenever cultures had been made.

Dr. CONNER said that two or three years ago he had presented an aneurism involving one of the sinuses of Valsalva, and producing a bulging forward of the conus of the pulmonary artery, and causing a very marked pulmonary stenosis, in which the appearances were strikingly similar to those of the present case. There appeared in the specimen just presented also to be a laminated clot.

Dr. LIBMAN said that the examination seemed to show that this was a developing abscess of the heart muscle.

#### EFFECTS OF REPEATED INTUBATION.

Dr. DAVID BOVAIRD made some remarks on this subject, and presented illustrative specimens. He said that in some instances it was necessary to practise intubation repeatedly on the same person. In one case occurring under his observation, a child had been intubated forty times, yet had recovered. The first specimen presented had been taken from a child about three years of age, who had been suddenly seized with a croupy cough and severe dyspnoea. Antitoxin had been given promptly, and intubation done almost immediately afterward. A few days after the tube had been removed, but recurring stenosis had required intubation again. While the tube was still in place, the child had developed measles and then pneumonia, and had died as a result of these complications. At the autopsy there had been found at the base of the epiglottis an ulcer of considerable size. In the body of the larynx, just within the cricoid ring was a large and deep ulcer. This ulcer involved the cricoid



cartilage, severing it completely in front. Farther down in the larynx and near the trachea were two small ulcers, corresponding to the lower end of the intubation tube. The ulcer at the base of the epiglottis represented the pressure made by the head of this tube. The ulcer at the cricoid cartilage was the result of the pressure of the body of the tube. The second specimen had been taken from a child aged six months. This child while recovering from measles had developed stenosis. A tube for a one-year-old child had been inserted, and the child had done well until the removal of the tube at the end of five days. Intubation had been required at the end of twenty-four hours, and this had to be repeated every two days for several days, and then almost constant re-intubation had been demanded because the child kept coughing out the tube. For this reason a tube for a two-year-old had been inserted, with like result. A tube for a three-year-old had then been introduced. This specimen showed large and deep ulcers at the base of the epiglottis. Nothing could be found representing the cricoid cartilage, it having apparently been absorbed by reason of pressure. There were three ulcers corresponding to the points of pressure of the lower end of the tube. In both specimens it was interesting to note the amount of injury done to the cricoid cartilage. As this cartilage was not only a support for the larynx, but the attachment of important muscles, it was evident that the destruction of this cartilage would be in itself sufficient to explain the recurrent stenosis of the larynx, the muscles by which the glottis might be opened no longer having a *point d'appui*.

#### CYSTIC KIDNEY.

Dr. BOVAIRD also presented a specimen of congenital cystic kidney removed from a child, aged one year, dead

of some enteric affection. The right kidney was the one involved. On the other side the pelvis of the kidney was found dilated and filled with gravel. There was also a stone of some size. These conditions on the left side suggested a possible explanation of the cystic degeneration on the right, that the ureter or pelvis of the kidney had been blocked by a stone and the cystic degeneration had resulted from the obstruction. No stone or gravel could, however, be found on the right side.

Dr. W. P. NORTHRUP said that these two cases, following closely after measles, suggested the explanation of the tissues breaking down so easily. These cases of so-called "retained intubation tube" were both of comparatively short duration. One should bear in mind the behavior of measles in a children's institution. Such children got up pressure necrosis on the back of the head, cancrum oris, necrotic processes on the fingers, and pressure sores on various parts of the body. Their tissues had diminished resisting power. As regarded the prolonged retention of intubation tubes, a case had just been reported in the *Archives of Pediatrics*, October, 1900, p. 737. In this case there had been subglottic stenosis, and after repeated intubations and extubations in the hands of a novice, the tissues had been denuded and had then grown together. Ultimately a tracheotomy had been required, and the child had died from pneumonia after this last operation. In this case the larynx had presented distinct hour-glass constriction and complete atresia.

PHOTOMICROGRAPHS OF FILARIA SANGUINIS MADE WITH  
A SPECIAL LENS.

Dr. BUXTON exhibited a series of photographs taken with a one-tenth Spencer lens from specimens mounted in balsam and examined through  $\alpha$ -brom-naphthalene

instead of oil of cedar, as recommended by Dr. Piffard. Certain new striations appeared in some of the photographs taken with this lens.

Dr. EWING said that the method employed had developed in the body of the filaria a peculiar cross striation, which was evidently part of the structure of the capsule of the filaria. So far as he knew it had not been described before, and certainly had not been observed in any previous drawing or photomicrograph.

#### EFFECTS OF MILK PRESERVED BY FORMALIN UPON MICE AND KITTENS.

Dr. WILLIAM H. PARK read a paper with this title. He said that the addition of formaldehyde to milk made the proteid material somewhat less digestible. The proportion required to preserve milk was about 1:50,000, and in this quantity digestion might be retarded about ten per cent. It was not contended that the end products were different, only that the digestive process was retarded. Certain reported investigations seemed to leave in doubt the question of the effect of formaldehyde on small animals. In one series small gains in weight were noted in kittens and rabbits, and a slight loss in a guinea-pig only. Another investigator had used three or four kittens for each control, and had experimented with different proportions of formaldehyde. When 1:50,000 formaldehyde was used, one of the kittens died in the third, and two in the fourth week, while the other two gained in weight. Of four kittens getting 1:25,000 formaldehyde, all gained. Of five getting 1:12,500, two died in the fourth week, two gained, and one lost. All the control kittens were reported to have gained—certainly a very remarkable showing. It had been stated that 1:50,000 retarded the increase in weight twenty-



nine per cent., and 1:12,500 sixty-nine per cent. These statistics had been manipulated in such a way as to leave grave doubts as to their reliability. Dr. Park said that he had accordingly experimented with young white rats, using 1:50,000, 1:10,000, and 1:1000 solutions of formalin. All of the rats had gained in weight and at very nearly the same rate. A series of experiments had then been undertaken with cats, using pure milk, milk containing 1:10,000, and milk containing 1:1000 formalin. At first they were fed on pure, selected milk. They all gained on this, but the 1:1000 much less than the 1:10,000, 1:50,000, or pure milk series. Later, on changing to ordinary milk, the cats lost weight, except those fed on milk containing 1:10,000 formalin. The explanation probably was that the ordinary milk in the hot weather (August) fermented through the night in the feeding-dishes, except the milk containing 1:10,000 or more formalin. Ordinary store milk in summer contained not only great numbers of bacteria, but also the products of their growth. He was inclined to think that among the poor, who could not keep the milk properly cold and sweet, it might be well during the hot months to introduce into the milk a small quantity of formalin, or if possible a better preservative, and label such milk "preserved milk," because it would do less harm in the milk than the products of fermentation.

#### *Discussion.*

Dr. NORTHRUP thought the question propounded by Dr. Park could have but one answer. If the formalin only retarded digestion, then it would certainly be better to feed this to children than to give them milk containing a large quantity of bacteria and all sorts of by-products. If it were simply formalin delaying slightly digestion,

against milk containing bacteria plus the never-to-be-forgotten toxins incidental to such growth, the answer was in favor of the lesser poison, namely, formalin. The great trouble was to accomplish this without opening the way for many abuses.

---

*Stated Meeting, November 14, 1900.*

EUGENE HODENPYL, M.D., PRESIDENT.

A CASE OF CONGENITAL DEFORMITY OF THE HEART.

Dr. N. B. POTTER reported this case, which occurred in a child supposed to have been perfectly well up to the age of two years. At this time there had been an attack of cholera infantum, and the attending physician announced that heart disease was also present. The speaker said that he had seen the child in a hospital last December, and at that time there had been marked clubbing of the fingers, associated with marked cyanosis and dyspnoea. There was also an otitis media on the left side. The child had, however, recovered, and had remained perfectly well up to October 1st. Symptoms of meningitis had then made their appearance, and death had occurred ten days later. The autopsy had revealed an abscess, the size of a large lemon, in the right frontal and parietal regions. There was a basilar meningitis. The cultures from the abscess and from the meninges were sterile. It was remarkable that such a large abscess could exist for so long a time without giving rise to symptoms. Examination of the heart showed the right ventricle to be nearly absent. The blood came from the vena cava through the foramen ovale into the left auricle, and from there passed to the left ventricle and thence to the aorta. The blood supply to the lungs came from a

branch of the aorta. A loud systolic murmur had been audible during life all over the pericardium without any particular spot of maximum intensity.

*Discussion.*

Dr. DAVID BOVAIRD said that this congenital anomaly of the heart was interesting, because of its peculiar association with abscess of the brain. This association had been noticed repeatedly, but so far no adequate explanation had been offered. This was the third case of the kind that he had seen.

A CASE OF RUPTURE OF THE SPLEEN; MALARIA.

Dr. POTTER also reported the following case: A sailor had been admitted to the hospital with a chill, and had died shortly afterward in spite of large doses of quinine. The organism of æstivo-autumnal fever had been found in his blood before death. At autopsy, a rupture of the spleen in the posterior and superior portions had been found. It was the kind of rupture seen as a result of violence, though there was no history of such traumatism. He did not think the rupture of the spleen had been the cause of death. Less than one pint of blood had been found in the abdominal cavity. The spleen weighed one pound six and a half ounces.

*Discussion.*

Dr. O. H. SCHULTZE reported several cases. The first was a case of apparent spontaneous rupture of the spleen occurring in a male adult suffering from malaria, reported by kind permission of Dr. E. J. Donlin. The deceased had been found dead in his room by the coroner's physician. From the fact that there had been well-marked post-mortem hypostasis in the frontal region, and in addition secondary hypostasis on the posterior aspect of the



body, he had been led to believe that an interval of eight or ten hours had elapsed from the time of the man's death, until he had been turned into the second position. About two quarts of fluid but also clotted blood had been found in the peritoneal cavity. The source of this hemorrhage was a laceration of the spleen. This organ looked like a mass of thick chocolate, and no portion could be absolutely identified as the spleen except a small shred of capsule. Some of this softened tissue had been examined microscopically, and identified as that of the spleen. It contained a considerable quantity of dark pigment. A smear from the bone marrow had been made, and was exhibited to the Society under the microscope. It showed quite a number of spheroidal bodies and pigment resembling melanin in the red blood cells. This person was a steward, forty-five years of age, who was said to have suffered from malaria for about three weeks. There were absolutely no external marks of traumatism. This, in itself, did not warrant a diagnosis of spontaneous rupture of the spleen, but from the appearance of the softened tissue and the blood in the region of the spleen, he felt warranted in looking upon this as a case of malaria with spontaneous rupture of the spleen.

Dr. Schultze also reported the following case: A man, thirty years of age, had been admitted to Bellevue Hospital on August 22d. He had complained of severe pain in the epigastrium, and of having vomited on the preceding day. The bowels had not moved for four or five days, and the diagnosis of his physician had been intestinal obstruction. On the following night the operation of colostomy had been done, and no peritonitis had been noted at that time. The man had died very suddenly and unexpectedly about twelve hours afterward. At the autopsy there had been found a considerable degree

of hemorrhagic pancreatitis with fat necrosis. The surface of the peritoneal fat showed throughout minute areas of fat necrosins. In some places this fat necrosis extended some distance below the surface of the tissue. The mistake in diagnosis was very pardonable, as the condition had often been confounded with intestinal obstruction.

Dr. SCHULTZE presented a third case. The subject was a man about forty-five years of age, who had been brought to Bellevue Hospital by the Hudson Street Hospital ambulance suffering from epigastric pain and vomiting. He stated that he had been pushed against a wagon and run over, but the autopsy had shown no evidence indicative of injury. He stated that at the Hudson Street Hospital he had been catheterized because of retention of urine, and that he had been bleeding since that time. There was some bleeding from the meatus, and on several occasions while in Bellevue Hospital it had been necessary to catheterize him. In the intervals of catheterization a small quantity of blood had been passed. It was said that the passage of the catheter had been very difficult, but there had been no infiltration of urine or any hemorrhage into the peritoneum. On the third day external urethrotomy had been performed, and the bladder had been explored digitally with such vigor as to cause laceration of the prostatic urethra. At autopsy, the bladder contained blood clot about two ounces in amount, but no blood had escaped through the wall. A large amount of fluid had been found in the peritoneal cavity, and examination had proved it to consist mostly of bile mixed with serum. There was absolutely no jaundice of the skin or of the conjunctiva. The fæces presented a clay color as in jaundice. It was evident that there had been a leakage of bile into the peritoneal cavity, and this was found to have come from the right hepatic duct close to the liver. In



the retroperitoneal tissue posterior to and below the pancreas there was a large mass to which the greater curvature of the stomach and the transverse colon were adherent. On separating this adhesion, the pancreas was exposed, and a large cavity containing fluid and decomposed tissue of a green color opened. On section, the pancreas was normal. Close examination seemed to show peri-pancreatic fat necrosis. Whether the leakage in the right hepatic duct was to be explained on some score of injury or whether the necrosis of the fat proceeded along the adipose tissue in the hilus had not been determined. Laceration of the hepatic duct, he thought, must be very rare. The connection of traumatism with this case was very indefinite, and rested solely on the patient's statement that he had never been run over.

Dr. WARREN COLEMAN spoke of the case of hemorrhagic pancreatitis. The patient had entered Bellevue Hospital while the speaker was acting as substitute for Dr. W. G. Thompson. The patient had been seized a day or two previously with vomiting and intense pain in the epigastric region, darting toward the left side, though on admission to the hospital the pain was located chiefly in the right hypogastric region. The diagnosis of the physician who sent him to the hospital was intestinal obstruction. There was some tenderness on the right side, and two of the physicians who saw him thought there was rigidity of the muscles. The physical examination, however, was especially difficult, because of the thickness of the abdominal walls. The patient's temperature was 103° F., and the pulse rate 140-150. The characters of the pulse were very significant—rapid, small, feeble, and thready—a condition of heart action not consonant with a temperature of 103° F. of only a few days' duration. These characters should have suggested pressure stimulation of the solar plexus, but did not at the time. In view of the



patient's condition and the possibility of the existence of appendicitis, operative interference was decided upon, and the case transferred to the surgical division of the hospital.

Dr. GEORGE P. BIGGS said that he had presented two similar cases to the Society some years ago. The symptoms had been almost identical with those just emphasized by Dr. Coleman. Both had been diagnosticated as appendicitis, and one had been operated upon. The great rapidity and weakness of the pulse had been noted in one of these cases, and also the presence of marked dyspnœa.

Dr. J. H. LARKIN asked if fat emboli had been looked for in the brain and in the other organs. The association of a sudden death with a fracture seemed to make such a question pertinent.

Dr. BROOKS said that the autopsy had been a complete one, and that a very careful and patient search had been made for fat emboli, but nothing of the kind had been found.

Dr. SCHULTZE asked what had been the condition of the lymphatic system.

Dr. BROOKS replied that there was no enlargement of the lymphatics, though they were looked for in every part of the body. The spleen was not much enlarged.

#### A CASE OF TUBERCULOUS ENDOMETRITIS, SALPINGITIS, AND PERITONITIS IN AN INFANT OF TWENTY MONTHS.

Dr. GEORGE P. BIGGS reported this case. Five months prior to death a painful swelling had appeared on the right side of the abdomen, accompanied by constipation and gradual emaciation. On admission to the New York Hospital, there was a large tender mass in the right side of the abdomen, thought to be an inoperable tumor of the kidney. Death occurred about one month later. At the

autopsy this tumor had been found to be a mass of fæces. The rest of the peritoneal cavity was protected by adhesions in which were many tubercles. An old perforation with rounded, smooth edges established free communication between the fecal cavity and the middle portion of the small intestine. There were a few characteristic tuberculous ulcers in the lower portion of the ileum. The mesenteric glands were enlarged and partially caseous. The involvement of the lungs was limited to a few recent miliary tubercles. The specimen presented consisted of the uterus and its appendages. The wall of the body of the uterus was 4 mm. in thickness, and its inner 2 mm. was distinctly caseous and showed small areas of hemorrhage. The appendages were embedded in a mass of adhesions containing many tubercles. The Fallopian tubes were enlarged and contained caseous material. Only the surface of the ovaries was involved. The case was apparently one of primary intestinal tuberculosis with perforation and the formation a large fecal cavity, the process in the genital cavity being secondary. The process in the uterus must have been a fairly old one, as shown by the extensive caseation.

#### CYST OF STOMACH.

Dr. SCHULTZE also presented a specimen of cyst of the stomach as a pathological curiosity. It had been found in a woman, about forty-five years of age, who had died from causes entirely independent of the stomach. The tumor projected about as much into the cavity of the stomach as on the peritoneal surface. It was situated about half-way between the greater and lesser curvature in the anterior part of the wall. On section, the tumor had been found to be embedded entirely in the layers of the muscularis. It resembled, somewhat, in the gross, a cyst aden-

oma, but microscopical examination had failed to confirm this. It contained only large blood-vessels, connective tissue, and considerable areas of a substance resembling mucin.

#### DEATH FROM ETHER ANÆSTHESIA.

Dr. HARLOW BROOKS reported this case. The patient was a man, thirty-six years of age, of good habits, and free from syphilitic taint. He had received a compound fracture of the forearm which exposed both the wrist and elbow joints. When picked up he had not been in a condition of shock, and had been seen by a surgeon about three hours afterward, at which time there had been no urgent symptoms. The intention had been to amputate the arm the next morning. The man had passed a good night, and about eleven o'clock the following morning had been prepared for the operation. No lesion of the heart and lungs had been found, and the urinary examination had been negative. Squibb's ether had been used, and it had been taken without much struggling through what is known as the "butcher-sleeve cone." Plenty of air had been admitted with the ether, and the pulse had improved under the first inhalations of ether. While preparing the arm, the pulse flagged, and for this reason a hypodermic injection of strychnine and whiskey had been given. About twenty minutes after the beginning of the anæsthetic surgical anæsthesia had been reached, and the amputation was begun by an incision through the skin. The conjunctival reflex had disappeared at this time, the voluntary muscles were flaccid, and there was every indication that the anæsthesia was complete. Just as the skin was incised, the respirations became noticeably slower and there was some retching. A drachm of fresh ether was given, and soon the respirations became more natural.



Five minutes after this, the respirations suddenly ceased at the end of full inspiration, and the heart action ceased two or three minutes later. There was no cyanosis, but the anæsthetic was at once stopped, the throat cleared out, the epiglottis pulled forward, and artificial respiration performed. Strychnine and ether were given hypodermically at first, and the limbs were elevated. Air could be forced into the lungs, but there was no spontaneous respiration. Death probably occurred about eight minutes after the first symptoms of danger had appeared. About four ounces of ether had been used. A very careful autopsy had been made. There was no indication of previous disease. There was no injection of the blood-vessels of the respiratory tract in any part. The lungs were not congested or œdematous. At the autopsy, which was done about three hours after death, there was no odor of ether about the lungs. The ventricles of the heart were contracted as one would naturally expect from the mode of death, although the right auricle was moderately dilated. There was a small calcareous nodule on the posterior surface of the left-hand flap of the mitral valve. The valves of the right heart were normal. The heart muscle showed a moderate fibroid myocarditis, which was most marked in the papillary muscle of the mitral valves. The intima of the arch and the intima of the coronary arteries were the seat of quite extensive arteriosclerosis. This sclerosis stopped short with the arch of the aorta. All the smaller vessels of the body seemed to be normal, even on microscopical examination. The connective-tissue increase was of the adult form, and there was no indication that the condition was a progressive one. He had come to the conclusion that the condition was a functional arteriosclerosis occurring in an athlete's heart. The kidneys were like fibroid kidneys, yet on section this connective-tissue increase was not so apparent. There was

only a slight connective-tissue increase of the adult form, and it was not progressive. The liver and all the other organs were normal. There was nothing distinctly abnormal in the central nervous system. A most noticeable thing in the nerve cells was an unusually plentiful deposit of coarse, brown pigment—a lesion not considered to be of special pathological significance. Careful examination of the ganglion cells by Nissl's method showed only slight and occasional degenerative lesions. The cells of the medulla were also normal. This careful examination precluded the possibility of an embolus or of discoverable lesion in the ganglion cells. The cause of death was apparently the result of the action of the anæsthetic upon the nerve cells; but, owing to the short duration of the etherization, it was hardly to be expected that any lesion would be produced which could be recognized after death by our present methods. Many surgeons were of the opinion that death from anæsthesia was most commonly the result of some obstruction in the respiratory passages, as for instance an accumulation of mucus. He had been surprised to find that most authorities agreed that the heart continued to beat for some time after respiration had ceased, when death occurred during etherization. The case presented was one of death from respiratory failure from the action of the ether on the respiratory centre, or possibly by poisoning of the respiratory muscles. The case was most instructive as showing that death from ether anæsthesia might occur in a person apparently perfectly healthy, and when the conditions were most favorable for etherization and for resuscitation.

A CASE OF TUBERCULOSIS OF THE BILE DUCTS AND SOLITARY TUBERCLES OF THE CEREBRUM.

Dr. BIGGS also reported this case. The patient was a girl of three years. One year before death the child had



had a sickness diagnosed by the attending physician as consumption. From this she apparently entirely recovered. Seven weeks before death the cough returned, and with it gradual loss of flesh. The physical signs were those of general bronchitis with scattered areas of broncho-pneumonia. The temperature did not go above  $101.5^{\circ}$  F. The day before death the patient had a convulsion with twitching of the entire left side of the body. At the autopsy, numerous tubercles were found in the kidneys, spleen, liver, lungs, and lymph nodes. The bronchi contained much muco-pus, and the lungs showed many areas of tuberculous broncho-pneumonia. In the brain there were two solitary tubercles, the larger of which measured  $2.3 \times 2 \times 2$  cm., and occupied the anterior portion of the left gyrus fornicatus. The smaller tubercle was in the left cuneate lobe. In addition to the distinct tubercles there were found throughout the liver many cysts 1 to 6 mm. in diameter, the contents of which were grumous material of greenish or yellow color. The gall bladder and larger bile ducts were normal. On microscopical examination the cyst walls showed typical tuberculous tissue. The presence of bile pigment and small remnants of mucous membrane proved the involvement of the bile ducts.

#### *Discussion.*

Dr. NORTHRUP asked what was the condition of the bronchial lymph nodes in the first case.

Dr. BIGGS replied that they were slightly enlarged, but were not tuberculous.

Dr. NORTHRUP said that in two hundred cases of tuberculosis in infants and small children, collected by Dr. Bovaird and himself, there had been just three in which the evidence that the process was oldest in the mesenteric lymph nodes was manifest. He had presented a



few specimens of tuberculous liver in infants in which it had been conspicuous that the bile ducts were very much thickened and dilated, and evidently the seat of tuberculosis. It did not seem to him that this class of lesions was unusual in infants.

#### A CASE OF DUODENAL ULCER OF EMBOLIC ORIGIN.

Dr. LEWIS A. CONNER presented a specimen taken from a man of about forty-five years of age, who had sustained a traumatic amputation of the middle third of one leg. There was such serious infection that amputation at the knee had been done by Dr. Stimson at the Hudson Street Hospital. This wound had showed gaseous decomposition, but after severe infection lasting several days the man had recovered from the operation. After an interval of several days he had suddenly vomited enormous quantities of blood, and had died within a few hours. The termination of the case had been startling and most unexpected. At the autopsy a thrombosis of the entire length of the femoral vein had been discovered. This had been concealed during life by the dressing. There was also an embolus in a large branch of left pulmonary artery which, curiously enough, had given rise to no symptoms. In the duodenum immediately below the pyloric ring was a recent ulcer 3 cm. in diameter, and projecting from the centre of the base of the ulcer was a minute embolic plug completely filling the lumen of a small twig of the superior pancreatico-duodenal branch of the hepatic artery. The case was of unusual clinical interest, for the embolus, small enough to pass through the pulmonary capillaries, had found lodgment in the duodenal mucous membrane. The very large gastric hemorrhage seemed to him rather unusual in a case of duodenal ulcer. Again, this ulcer must have developed

rather rapidly, because from the time of the accident to death was not more than three weeks. This ulcer must have formed and gone through the wall of the duodenum in the course of a few days. This was the second or third case of gastric or duodenal ulcer in which he had found an occluded artery in the base of the ulcer.

#### A CASE OF LARGE CARCINOMA OF THE PROSTATE.

Dr. CONNER also presented a soft carcinomatous tumor of the prostate taken from a man of seventy, who had died within a few hours after entering the hospital. He had presented symptoms of uræmia. A large, elastic tumor had been found in the right iliac region. At the autopsy the whole pelvis had been found filled with an encapsulated, lobulated elastic tumor, measuring  $15 \times 12 \times 11$  cm. The bladder had been lifted up out of the pelvis, and its mucous membrane was not involved in the growth. The rectum also had escaped. The kidneys were much diseased, and one ureter was dilated as a result of obstruction.

#### *Discussion.*

Dr. C. A. HERTER said, with reference to thrombosed vessels in ulcers of the duodenum, that he did not think it at all uncommon to meet with hyaline thrombosis in vessels of ulcers of the stomach. He had seen these a number of times, and they seemed to play an important part in the formation of the ulcer.

Dr. I. LEVIN asked if there had been a complete obstruction of the urethra, and if the case was of uræmia from obstruction, or of coma connected with carcinoma.

Dr. CONNER said that the kidneys showed that death had been unquestionably due to uræmia. There had not been retention of urine, and little or no obstruction

in the urethra. The large mass in the right iliac fossa was an encapsulated metastasis. Metastatic growths were found along the retroperitoneal glands and in the bronchial lymph nodes.

#### A CASE OF DIABETIC COMA.

Dr. C. A. HERTER made some remarks on this case, which he had seen only at autopsy. The patient was a well-nourished woman, about sixty years of age, who had entered the hospital in a stuporous state. She had soon become comatose. The urine contained a large amount of sugar. The case having been supposed to be one of diabetic coma, a quantity of sodium bicarbonate was infused into the venous system. The patient rallied slightly, but soon became comatose, and died within twenty-four hours. At the autopsy the brain showed considerable œdema. There was no thickening of the pia or any of the ordinary indications of chronic alcoholism. The lungs were normal. The heart muscle was pale. The liver weighed 1617 gm., and was moderately fatty. The bile ducts were pervious. The pancreas was soft and very small, weighing only about half an ounce. The spleen was of medium size and appeared normal. The kidneys were large and pale, and the cortex was somewhat fatty. The suprarenals were normal. The urine in the bladder contained many granular and hyaline casts. The urine was examined with a view to ascertain whether all of the bases were neutralized by the known acids of the urine. If the bases were in excess, this excess must be due to the presence of organic acids in the urine, which had served to neutralize the bases. The blood and the cerebro-spinal fluid were also examined. The simple atrophy of the pancreas was interesting. Of forty cases reported by Hansemann in which pancreatic



disease coincided with diabetes, thirty-six were cases of simple atrophy of the pancreas. Dr. Herter said that his experience had been that simple atrophy was extremely common in diabetes. The proteids of the blood were removed by means of alcohol and the filtrate treated with Fehling's solution. The result showed only a very small quantity of sugar. It had been thought for many years that the blood contained normally 0.1 to 0.3 per cent. of sugar, but these observations of Claude Bernard had been recently called in question, the reducing substance in the blood not being thought to be sugar but a substance called jecorin. It was probably an actual compound of dextrose with lecithin. If this theory were correct, it would explain why the blood normally contained no sugar, or only an exceedingly small percentage. The cerebro-spinal fluid showed more reducing substance than the blood, but it was not entirely clear that this substance was sugar. The urine contained 16.73 per cent. of the nitrogen of ammonia. This ammonia usually runs from 1 to 4 or 5 per cent. of the total nitrogen, varying somewhat with the diet. Such a high percentage of the nitrogen of ammonia, the urine not having undergone decomposition, indicated that the ammonia intended, under normal conditions, for synthesis with carbonic acid for the formation of urea, was diverted by the acids present, and was used for the neutralization of these acids. If this did not occur, death in coma would take place at a much earlier stage of diabetes. It was only when the quantity of acid was so great that the ammonia could not effect a neutralization that the acid took out the sodium, lessened the acidity of the blood, and led to coma. It was calculated that 10 gm. of oxybutyric acid, and 33 gm. of sugar were excreted in twenty-four hours, thus making an enormous proportion of oxybutyric acid for the quantity of sugar

present. In looking over other cases that he had studied he found in one that there had been 20 per cent. nitrogen of ammonia and 25 gm. oxybutyric acid in twenty-four hours about one month before the patient went into coma, and the day before coma 21 per cent. of the nitrogen of ammonia and 23 gm. of oxybutyric acid. In a second case of coma the nitrogen of ammonia was 20.88 per cent. and the oxybutyric acid 27 gm. He had studied a few cases of diabetes in which coma had not occurred. One of these cases was now in Bellevue Hospital under Dr. Loomis's care. There were 27 per cent. of the nitrogen of ammonia and 24 gm. oxybutyric acid, and this had led him to give a bad prognosis, and on investigation he had found that the patient had been stuporous for the past few days. In another severe case there had been 16 gm. of oxybutyric acid and 20 per cent. of the nitrogen of ammonia. In still another case there had been 13 gm. of oxybutyric acid and 16 per cent. of nitrogen of ammonia. By changing the diet the nitrogen of ammonia had fallen to 11 per cent. and the oxybutyric acid to 8 gm. He had now under observation a case of diabetes that had lasted for fifteen years. At present there was about 5 per cent. of sugar in the urine. It contained 3.62 per cent. of nitrogen of ammonia and 1.8 gm. of oxybutyric acid. As there had been only a slight change in some months, the outlook was fairly good. Whenever the nitrogen of ammonia reached the neighborhood of 20 per cent. one must look out for coma. It was, however, a far less reliable index than the balancing of the acids and bases. This unreliability was particularly noticeable when the acid excreted was comparatively small in amount.

Dr. CONNER asked if diacetic acid was no longer considered to be of importance in these cases.

Dr. HERTER replied that he had said nothing about

diacetic acid, because it was only one of the acids, and probably seldom amounted to more than 10 per cent. of the acids present. Unless the urine was very fresh it was impossible to study this acid. He believed beta-oxybutyric acid was the chief acid.

PRIMARY CARCINOMA OF THE LUNG WITH MULTIPLE  
METASTASES.

Dr. E. HODENPYL presented specimens from this case. The patient was a man, fifty-five years old, who had been previously well. He had been ill for five months with cough, dyspnoea, and pain in the left chest before coming under observation. There was dulness over the upper lobe of left lung with very distant bronchial breathing and absence of voice and vocal fremitus. Puncture of the pleural cavity and examination of the sputum had proved negative. A diagnosis of probable tumor of the lung was made. The man had had two or three slight hæmoptyses. During the last month he had become delirious. At the autopsy a new growth had been found in the left lung. The costal pleura on that side was very greatly thickened, though not involved in the new growth. There was also a fairly acute pericarditis. The suprarenal capsules had been replaced by a new growth, and the pancreas was studded with large nodules. There was a new growth in the right occipital lobe of the brain. The other lung was entirely normal. Microscopical examination showed the growth to be a large alveolar carcinoma.

Dr. F. C. Wood then read a paper on

ENDOTHELIOMA.

He also exhibited a patient. This man had had a tumor in the parotid region for about five years. It had



not grown rapidly only during the past few months. The tumor was adherent to the deep fascia, and was cartilaginous. The object of the paper was to show that there was a class of mixed tumors characterized by slow growth for a long time, and then by a more rapid growth and a change in structure coincident with the change to malignancy. These tumors were derived from the endothelium of the lymph spaces. The exact embryological position of the lining endothelium of the lymph spaces is as yet unknown, but the general assumption among embryologists is that it is derived from the mesoderm. The morphology of the tumors derived from the endothelium of the lymph spaces and their separation from epithelial structures classes them with the sarcomata as a special sub-group. Until the final decision as to the true embryological origin of the endothelium can be furnished, it is most simple to avoid the question of embryology entirely, and to classify such tumors according to their origin from the tissues as finally differentiated at the time of birth. A sharp distinction should be drawn between the endotheliomata arising from the parotid and sub-maxillary region and those arising from the lymph spaces underlying the peritoneal endothelium. The character and morphology of the two growths is entirely different.

A number of microscopical specimens were then projected on the screen by means of an electric lantern.

---

*Stated Meeting, December 12, 1900.*

EUGENE HODENPYL, M.D., PRESIDENT.

ROUND-CELL SARCOMA OF THE THIGH.

Dr. F. R. BAILEY presented a tumor of the thigh that had been removed by amputation. The patient, a woman, aged twenty-two years, was first seen on July 7, 1900.

.

The family history was entirely negative, as was also the personal history in so far as it has bearing upon the present condition. The patient complained of a feeling of slight stiffness in the right thigh, but physical examination was negative. About one week later a second examination was made. There was no pain or tenderness, but on the outer side of the right thigh there was a scarcely perceptible swelling. From this time the swelling slowly increased; the patient began to complain of a dull aching; there was slight tenderness on deep pressure. About the 1st of September an acute condition set in with heat, redness, and a temperature ranging from 100° to 103° F. At this time the swelling presented every appearance of an abscess. These symptoms subsided in about ten days. There remained no pain, but slight stiffness and swelling. The patient went about as usual. The condition was now about the same as when the patient was first seen. The last of October there was a return of pain and tenderness with a rapid increase in the swelling, but without inflammatory symptoms. An exploratory incision was made on November 3d. On incision of the deep muscles and fascia a semi-fluid transparent substance oozed out. Sections from this were exhibited under the microscope. They showed the growth to be a sarcoma of the small round-cell variety. On November 15th, a hip-joint amputation was performed by Dr. Marvle. Flaps were cut as short as possible and apparently through healthy tissue. Slight force, used in attempting to rotate the femur during operation, resulted in its fracture, as seen in the specimen. The upper part of the femur had been sawed longitudinally and showed the condition of the interior. The subsequent history of the patient had been uneventful. The wound was entirely healed with the exception of a small area at the point of insertion of the drainage tube.

## ANEURISM OF THE THORACIC AORTA.

Dr. THEODORE C. JANEWAY reported the following case, which had been under his observation in the University Medical College clinic from June, 1900. The patient was a Frenchman, forty-one years of age, a glass-blower by occupation, who had been exposed to the fumes of lead. There was a history of probable chancre seventeen years before, without definite secondary symptoms. He had been seen for the first time last July. Three years before this he had been taken with pain in the upper part of the abdomen, and this had been pretty constant since that time, and had slowly increased in intensity. He said the pain had always been worse when he was lying on his back. The abdomen was tender in the epigastrium. Examination of the spine had been negative, and jarring had caused no pain. There was a slight area of dulness in the left hypogastric region, which pulsated very slightly. After two or three examinations the diagnosis of a small abdominal aneurism had been made. There were no thrill and no murmur, and the heart was not enlarged. No signs could be found over the back on repeated examination. There was a slight first and second sound to be heard over the tumor. The man had been admitted to Bellevue Hospital in October. While there he had developed a systolic thrill and murmur over this mass, and also over the heart. He had been found dead in the water-closet one morning. At the autopsy a large quantity of blood had been taken from the left pleural cavity, and an aneurism, apparently of the abdominal aorta, found ruptured in it. A careful examination of the specimen in the laboratory showed that the diaphragm ran down over the tumor mass. Apparently the tendon of the diaphragm made the lowermost portion of the aneurismal sac, and there was no involvement of the



coeliac axis by the aneurism, so that it was probable that the aneurism was not of the abdominal aorta. The aneurism seemed to have been given off from the anterior wall of the last portion of the thoracic aorta and grown downward, carrying the tendon of the diaphragm with it. The fact that the aneurism had been held by the tendon of the diaphragm probably explained the very slow increase in size. By dissection through the upper portion of the sac and through the subpleural sac there had resulted a rupture into the pleura. Even at the autopsy the tumor had been taken for an abdominal aneurism.

#### HORSESHOE KIDNEY.

Dr. A. E. THAYER presented three specimens. The first was a good example of the horseshoe kidney, not discovered during life. The subject was a man of fifty years, who had a varicose ulcer on the right side of the leg. He had died of embolic abscess of the lungs. The specimen weighed 1.3 kgm., and measured, the right, 21 cm. long by 9.75 cm. broad by 7.25 cm. thick; the left, 17 cm. long by 7.5 cm. broad by 5.25 cm. thick. There was a large union of renal tissue at the bases of the kidneys. The pelves were displaced forward. The right renal artery came off low down, close to the junction, and sent a branch to the left kidney. There was one ureter common to both kidneys. There was some parenchymatous nephritis in the left kidney, and in the right were numerous cavities containing a considerable quantity of yellow puriform fluid. There was also a large stone in the pelvis of this kidney.

#### INTERSTITIAL PANCREATITIS AND FAT NECROSIS.

The second specimen had been taken from a German, forty-five years of age, who had been in the hospital from

October 3d to 26th. There had been a rather obscure train of symptoms, and the diagnosis had rested between abscess of the liver and empyema. There had been no fever and no glycosuria, and very little pain. When the body was opened there had been found general recent peritonitis, and the peritoneal cavity contained about 2000 c.c. of red purulent fluid. There was gangrene of the lungs. In the abdomen was a large fluctuating mass behind the peritoneum. When this was opened a large quantity of fluid and clotted blood and pus escaped. A small portion of the tail of the pancreas was found wedged in between blood clots. There were areas of fat necrosis over the mesentery. Some of the mesenteric glands were enlarged and cheesy. Microscopical examination showed interstitial pancreatitis and some fat necrosis, but no fat crystals. Smears had been made from the fresh lung, but no tubercle bacilli or other characteristic bacilli had been found. Cultures had also been made, but they had proved negative on ordinary media. By a modified Weigert method a few areas had been found in the tissues which looked a little like a streptothrix invasion. He thought the two conditions were probably coincident.

#### ARTHRITIS DEFORMANS.

The third specimen was a good illustration of arthritis deformans. The patient had shown multiple dislocations of the joints. The body was extremely emaciated, the total weight post-mortem being about twenty-eight pounds. The patient had been bedridden for twenty-six years. There was firm bony ankylosis of the right hip nearly at a right angle. The stomach showed a number of dark areas surrounded by a hyaline zone—apparently submucous hemorrhages. This woman had developed an acute gonorrhœal conjunctivitis soon after admission

to the hospital, and the source of this infection had not been discovered until the autopsy had revealed an enlarged uterus filled with a thick fluid of light green color which contained gonococci.

*Discussion.*

Dr. JAMES EWING thought the second case one of very great interest because cases of pulmonary lesions which are not tuberculous and yet resemble tuberculosis were not at all uncommon. He had been fortunate enough to see the cases published by Drs. Larkin and Norris. The particular features of the present case were the lesions in the lungs, the multiple gangrenous foci, and the peculiar caseous character of the lesions in the peritoneum and in the neighboring lymph nodes. When stained, they had failed to show tubercle bacilli, and cultures had also been negative. There had been since then one other probable case of streptothrix pneumonia, but no positive results had been obtained from cultures.

A METHOD OF OBTAINING BLOOD FOR CHEMICAL EXAMINATION.

Dr. J. S. THACHER made some remarks on this subject. He said that his object was to facilitate the making of routine chemical clinical examinations of the blood. He had made use of a flask having a tubule at the bottom ground to fit an aspirator needle. A bandage was tied around the upper arm to congest the veins at the bend of the elbow. The skin was disinfected, and then the needle attached to this flask was plunged into the vein, care being taken not to allow the needle completely to traverse the vein. Two ounces of blood were enough for a urea determination, and this quantity could be readily obtained with but little discomfort to the patient and almost no inconvenience to the physician. The



suction was made by the use of a rubber mouth-tube attached to the cork in the mouth of the flask. This study of urea in the blood had already proved extremely interesting. It was very important that the needle should be clean and also perfectly smooth, so as not to cause coagulation within the vessel. Dr. Thacher then gave a demonstration upon himself of the method of obtaining samples of blood with this apparatus.

#### REMARKS ON EMBEDDING IN CLOVE-OIL CELLOIDIN.

Dr. E. HODENPYL made some remarks on the use of a solution of celloidin in oil of cloves and ether as recently advocated by E. M. Stepanow (*Zeitschrift für wissenschaftliche Mikroskopie*, October, 1900, pp. 185-191). The advantages claimed for the method were : (1) It was quite as simple as the use of the ordinary solution of celloidin in alcohol and ether. (2) Impregnation went on rapidly, and was so complete that very thin section—3  $\mu$ —could be made. (3) It was applicable for double embedding in celloidin and paraffin. (4) It was especially useful for the central nervous system. Dr. Hodenpyl was induced to try the method on account of the rapidity with which material could be embedded and cut into sections, and he showed sections under the microscope which had been embedded in the clove-oil celloidin for but two hours, which were quite as thin and which stained quite as well as sections embedded in the ordinary way. The following was the composition of the solution recommended: Celloidin, 1.5 gm.; oil of cloves, 5 c.c.; ether, 20 c.c.; absolute alcohol, 1 c.c. It required about two days to make the solution. When finished it was of a lemon-yellow color. The technique of the method was quite simple. The soaking in alcohol and ether was entirely done away with. The specimen could be put

directly into the solution of celloidin, from strong alcohol, and after it had remained there two hours or more it could be blocked, hardened partially in the air, and finally in eighty per. cent. alcohol. A much quicker way was to immerse the block in pure chloroform; or, after embedding in celloidin, the specimen could be dropped in benzol for a short time and then a frozen section made. Very much thinner sections could be made if the specimens were first soaked for half an hour in oil of cloves, and then embedded in the clove-oil celloidin.

THE USE OF THE AQUEOUS EXTRACT OF THE SUPRARENAL  
CAPSULE AS A HÆMOSTATIC.

Dr. W. H. BATES exhibited a preparation of the active principle of the gland of the strength of one part in ten thousand. He also showed three or four other alkaloidal preparations. He said that the aqueous extract of the suprarenal gland was the most powerful known hæmostatic. The effect depended much upon the preparation employed. One part of the dried gland should be mixed with ten parts of water in the form of an emulsion. When prepared immediately before using, this emulsion was decidedly active, but it might lose its efficacy in a few hours. An alkaloid had been obtained from the suprarenal extract by Takamini, which he had named adrenaline. Its keeping qualities were good when sterilized and protected from the air. In controlling hemorrhage from the lungs, stomach, bladder, or other organs, the dried, powdered gland was efficient; tablets were less efficient because the excipient interfered with its action. The extract must be brought in contact with the vessels, and a bloodless operation could be done by frequently repeated applications of the extract. Operations on the skin had been made bloodless by the hypodermic use of the ex-

tract. Pain neutralized the effect of the suprarenal extract almost immediately. It seemed to be a specific in hæmophilia. The extract had been used successfully in connection with packing of the uterus to control post-partum hemorrhage. Hæmoptysis could be controlled by directing the patient to chew five to ten grains of the suprarenal extract, and swallow it without water at short intervals. The speaker drew the following conclusions: (1) suprarenal extract was the most powerful hæmostatic known; (2) hemorrhage from mucous membranes could be always controlled by its use; and (3) when it controlled hemorrhage, locally or by internal administration, it did so in less than one minute.

---

*Anniversary Meeting, January 9, 1901.*

EUGENE HODENPYL, M.D., PRESIDENT.

DIFFUSE GANGRENE OF THE LUNG.

Dr. LEON T. LEWALD presented several specimens. The first one was from a case of diffused gangrene of the lung. Nearly the whole of the lower lobe was found to consist of a soft greenish mass. Rupture had occurred into the pleural cavity, and the latter contained several ounces of dark greenish fluid. The patient was a man twenty-six years of age, who had been admitted to Bellevue Hospital on December 19th. On admission, a few moist râles had been found over both lungs, and his temperature had been 103° F. A diagnosis of bronchitis was made. The urine contained a trace of albumin, had a specific gravity of 1.020, and was free from sugar. The next day the temperature was 99° but quickly rose to 104°. On the third day the man had a chill, with a temperature of 105.4°. Eight hours later it had dropped to



98°, and again rose to 103° after a second chill. On the fourth day the sputum became offensive. The diagnosis of gangrene of the lung was made. Dulness was present over the upper portion of the lower lobe of the left lung. The temperature remained at about 104° for the next four days. Death occurred on December 26th. Examination of the blood at the time of admission showed a leucocyte count of 7650. On December 20th the leucocyte count had been 22,000. Aside from the gangrene of the lung the autopsy showed evidence of septic infection. The spleen weighed two pounds two ounces, and was very soft and pale. The liver and kidneys showed parenchymatous degeneration. The lung was stained with eosin and methylene blue, but no cultures were made.

#### DEATH FROM LODGMENT OF A PLUG OF TOBACCO IN THE BRONCHI.

The second case was one in which death had resulted from the inhalation of a large piece of tobacco. A man, twenty-five years of age, had been found dead in a stable. The autopsy showed signs of asphyxiation. On removal of the lungs the piece of tobacco was found completely blocking both bronchi and extending up into the trachea.

#### A CASE OF THROMBOSIS OF THE SUBCLAVIAN ARTERY.

The third case showed thrombosis of the left subclavian artery at a distance of one inch from the aorta. This had resulted in a dry gangrene of the arm extending up as far as the middle of the upper arm. The clot was formed on an isolated atheromatous patch. The patient was a man fifty-five years of age, and death had resulted from a septic broncho-pneumonia.

Dr. HODENPYL remarked that the peculiar odor of this

lung would lead one to suspect actinomycosis, but there was nothing else about the specimen to suggest this.

#### ADVANCED FATTY CHANGE OF THE HEART.

Dr. E. K. DUNHAM presented specimens from a case of advanced fatty change of the heart. The heart-muscle fibres from this case contained a great amount of fat. He had studied the case carefully to determine the relative amount of fat to the cell body. A diagram was shown, drawn to scale, of the heart-muscle fibre, the portions representing the fat drops having been cut out and weighed. It had been found in this way that about one-third of the area of the cell section consisted of fat. Measurements went to show that the area of the cell section was two and a half to three times that of the normal cardiac muscle fibre, so that the increase in the size of the cell was greater than the addition of fat. This raised the question as to whether this might not be an infiltration and not a degeneration. From these measurements there was certainly no reason for supposing that the fat was due to degeneration; on the other hand, fatty infiltration seemed quite a reasonable explanation in the light of these measurements. It was agreed that fat might be derived from proteids, because it was known that carbohydrates could be so derived. But the carbohydrates were so much more easily burned that it was probable that a large quantity of the fat in such cases represented a food-residue not readily oxidized and, in this sense, to be regarded as an infiltration. The case was one of very marked anæmia supposed, before death, to be pernicious anæmia, occurring in a person of forty years of age, but might have been one of secondary anæmia, due to advanced interstitial nephritis. The accumulation of fat in these heart-muscle fibres was probably the result of a

failure to burn the fat readily owing to the anæmic condition. Three sections from this case were exhibited under the microscope.

#### CONGENITAL ABSENCE OF THE KIDNEY.

Dr. OTTO H. SCHULTZE presented specimens from a man about thirty years of age, of whose clinical history nothing was known. The cause of death had been clearly shown to be an acute lobar pneumonia involving the upper and lower lobe of the right lung. The point of interest was the absence of the left kidney. A little above the level of the sacro-iliac synchondrosis was a vestige which was taken to be the "anlage" of the kidney and ureter. By this term was meant not only the site but the actual tissue which should have developed into a given organ. This vestige consisted of a canal in which there was a distinct bend. The upper portion was dilated into an ampulla composed largely of connective tissue with here and there a canal. Under the microscope this canal resembled closely a well-developed ureter. Above this enlargement were three canals branching off and passing externally to the ampulla. They were about 6, 5, and 3 cm. in length respectively. On cross section a number of canals could be found, and these were lined with cuboidal epithelium. The contents consisted of dark brown material which was shown under the microscope to consist of epithelial detritus. This duct he took to be the ureter, and the division as having taken place before the formation of the kidney. Surrounding this tube a fascia or sheath, passing from the brim of the pelvis into the true pelvis and inguinal canal, apparently connected with the tunica propria of the testicle. The testicle and epididymis were present, but they did not occupy their normal relations, the epididymis being stretched out above the testis. The tunica vaginalis testis passed upward and



included the tail of the epididymis. From the latter a very thin duct could be traced upward; it was apparently the vas deferens, but was lost at the bladder wall. On dissecting up the peritoneum the vas deferens and vesicula seminalis with the ejaculatory duct were seen on the right side. The seminal vesicle on the left side was not nearly so well developed. The orifice of the right ureter was quite distinct; that of the left ureter could not be found, nor could any trace of the left ureter be discovered at the bladder. This was because the left ureter passed upward upon the bend mentioned, becoming atrophic. Sections of the ureter had been made in various places, and of the right and markedly hypertrophied kidney. The size of this kidney was apparently due to compensatory hypertrophy.

Dr. DUNHAM asked if the recurrent portion of this ureter had been examined microscopically.

Dr. SHULTZE said that this portion had not as yet been examined.

#### CASES OF PRIMARY MALIGNANT NEOPLASM OF THE LUNG AND PLEURA.

Dr. I ADLER presented specimens taken from a man twenty-five years of age, who had entered the hospital with symptoms of obstruction of the upper part of the cava. Soon afterward symptoms indicative of obstruction of the lower part of the cava also were developed. There was a large area of flatness over the right chest. The heart was displaced toward the left, and puncture showed no fluid. A diagnosis of primary malignant neoplasm of the right pleura had been made. The man had been absolutely well up to a few weeks before coming to the hospital, yet death had occurred about two weeks after admission. The autopsy had verified the diagnosis of

such a tumor involving the mediastinum and causing pressure on the cava. The tumor peeled off quite readily from the sternum, leaving intact bone and fibrous tissue beneath. The tumor mass filled the anterior portion of the chest, and the lung was compressed upward and backward toward the spine. The diaphragm was displaced downward and the anterior mediastinum was filled with these masses. The upper cava was almost completely obstructed by the pressure of the growth ; the lower cava was obstructed but not to the same extent. The left lung was normal. With the exception of a few secondary deposits in the liver this organ was normal.

Dr. Adler presented a specimen from a second case of this kind. It had been taken from an old man who had been admitted to the hospital with a diagnosis of pulmonary consumption. He had not seen the man during life. At the autopsy the upper portion of the left lung was found to be made up of a tumor mass of soft and friable nature, and involving the pleura. Toward the apex the lung was more or less infiltrated with tumor substance. The lower lobe was nearly free, and the right lung was completely free. On section of the lung the invading mass was shown not to be fibrous but distinctly tumor tissue.

The third case presented had come under observation only a few weeks ago. The patient was a man about sixty-seven years of age, who had been well up to two months before admission to the hospital. At that time he had begun to fail in strength, and complained of some pain in the right chest and of difficulty in breathing. He was extremely cachectic and feeble. There was complete dulness of the anterior portion of the right thorax; the heart was displaced to the left and its action was very feeble. There was absolute flatness over the anterior part of the chest. There was an area of tympanitic percussion



note with lung resonance in the upper portion of the right thorax, and an area of well-marked dulness about half an inch wide extending across the chest, and again an area of tympanitic resonance, and again an area of absolute flatness. Here auscultation revealed no respiratory sound. The area of dulness gave diminished respiration with a few râles and considerable friction. Anteriorly there was no respiratory murmur and no vocal fremitus. It had been assumed that there was fluid in the chest, chiefly low down posteriorly, and that the area of dulness between the tympanitic areas was due to tumor infiltration originating in the pleura. In addition, it was thought that there were metastatic deposits in the lung itself and some hemorrhagic effusion. The autopsy confirmed this diagnosis. Neither the aorta nor the heart was affected by the surrounding mass of new growth. There were a few secondary deposits in the liver and in the right kidney. The left adrenal had been converted into a huge mass of tumor tissue.

The fourth specimen presented by Dr. Adler had been taken from a man whom he had seen quite frequently for a number of years in the practice of Dr. Lewengood. The man had appeared exceedingly healthy but was annoyed by a persistent cough and by some pain in the left apex. Examination had shown absolute dulness at this apex with absence of respiratory and vocal sounds. There was also bloody expectoration, and this had been repeatedly examined microscopically but with negative result. A diagnosis of tumor had been made quite early because this dulness had crept steadily backward. Death had occurred from a sudden and profuse hemorrhage. The autopsy had revealed a very thick pleura, which the microscope showed to be due to tumor tissue. There was considerable tumor tissue around the pericardium and the latter was very much thickened. Some of the tumor



tissue grew into the heart. There were a few small metastatic deposits in the liver, the kidney, and the bronchial glands.

Commenting upon these four cases, Dr. Adler said that all of them had presented the same features under the microscope. These were illustrated by several drawings. There had been considerable discussion on endothelium and epithelium, and their very similar structure. Waldeyer was inclined now to call the covering of the serous membranes epithelium, but it would seem that the name endothelium should be restricted to the lining of the lymph channels, the covering of the blood-vessels, and the lining of the blood capillaries. His own studies had led him to this conclusion long ago. When he said that these tumors were endotheliomata he had meant that they originated from the pleura alone, and that the cellular portions had originated in every one of these cases from the lining of the lymph channels. These growths had not originated from the epithelium of the free surface of the pleura or from the tissue of the lung. Wherever the lung in these cases was seen affected the involvement was secondary. From the studies that he had been able to make and from a careful examination of the literature he was inclined to believe that primary tumors of the lung originated only from the bronchial tract, from the epithelium of the bronchial surface, and especially from the epithelium of the bronchial mucous glands. They were in every case carcinomata. All the other tumors which did not originate from the bronchial surface or from the bronchial mucous glands originated from the pleura, and were all endotheliomata in the sense already mentioned. As such this must be classed among the fibrous-tissue tumors, and in close proximity with the sarcomata. The alveolar portion of the lung had thus far not been shown to be the seat of a primary tumor.

## CEREBELLAR ABSCESS; RUPTURE INTO THE FOURTH VENTRICLE.

Dr. GEORGE P. BIGGS presented a specimen of cerebellar abscess taken from a man forty-one years of age, who gave a previous history of defective hearing since the age of twelve years, following an attack of scarlet fever. Discharge from both ears had occurred at intervals, the last time being two months before admission to the hospital. Eleven days before admission he had been seized with severe pain in the back of the head, with vomiting and with partial paralysis of the right side of the face. His chief complaint was headache. On admission he was apathetic and in poor general condition. There were paralysis and impaired sensation and touch on the right side of the face, and the tongue deviated to the left. The left pupil was larger than the right, and both reacted to light. The neck was rigid and tender. There were nystagmus and conjunctivitis of the right eye. The pain in the head continued to be severe, and was associated with delirium. Death occurred three weeks after the onset of the acute symptoms. During the last few days of life hiccough was very persistent. The temperature varied from  $100^{\circ}$  to  $103.6^{\circ}$  and the pulse between 90 and 105. At the autopsy a meningitis was found at the base, and particularly over the right lobe of the cerebellum. Around the seventh and eighth cranial nerves on the right side was considerable purulent exudate, and these two nerves were distinctly softened. The lateral ventricles were moderately distended with sero-purulent fluid, as was also the third ventricle. In the fourth ventricle, which was considerably distended, were some offensive pus, and a pyramidal-shaped mass of necrotic material measuring 2 cm. in length and 1.5 by 1 cm. at the base. This mass of necrotic tissue had originated in an abscess in



the right cerebellar lobe close to the pons. A communication had been established between the fourth ventricle and this cavity by a rounded opening measuring 1.5 by 1 cm. The abscess cavity was pyramidal in shape with the base looking backward toward the ventricle, and its apex forward toward the middle peduncle. There was also a small perforation at the anterior extremity of the abscess cavity. The cavity measured 3 cm. antero-posteriorly, 2 cm. horizontally, and 1.5 cm. vertically. The floor of the fourth ventricle was covered with a thick layer of greenish and offensive pus. The petrous portion of both temporal bones was thickened, and on the right side was a quantity of offensive pus. The smooth rounded opening between the abscess cavity and the fourth ventricle suggested a process of some duration.

#### SPLENIC TUBERCULOSIS OF UNUSUAL TYPE.

Dr. BIGGS also presented specimens from a case of general tuberculosis, occurring in a colored child of nine years. The interesting feature was the tuberculosis of the spleen, which was very extensive and was characterized by firm, gray tuberculous nodules of unusually large size, having the appearance on first inspection of tumors. The spleen weighed 280 gm., and the nodules were  $\frac{1}{5}$  to  $1\frac{3}{4}$  cm. in diameter. Microscopical examination showed extensive caseation, and but very few giant cells. In other parts of the body the tubercles also showed a tendency to grow to an unusual size, and to be the seat of extensive caseation.

A second specimen exhibited was from a colored man fifty-three years of age, with a history of tuberculosis extending over five years. The tuberculous process was quite general throughout the body. The spleen was very large and intimately adherent to the surrounding tissues.



It weighed 1010 gm. In this spleen were very large groups of miliary tubercles very closely aggregated, forming firm grayish areas  $\frac{1}{2}$  to 2 cm. in diameter. Microscopically these areas showed the usual structure of miliary tubercles with many giant cells, a pronounced tendency to the formation of fibrous tissue, and but slight coagulation necrosis. The same features were noted in the tuberculous lesions of other parts of the body.

*Discussion.*

Dr. THEODORE C. JANEWAY asked if there was any record to show how long before death the enlarged spleen had existed.

Dr. BIGGS replied that both cases had been in the hospital only a few days, and no record on this point had been obtained. In both cases the enlargement of the spleen had been easily made out on physical examination.

TWO CASES OF BACTERIAL INFECTION OF THE URINARY TRACT.

Drs. A. W. WILLIAMS and R. J. WILSON reported these cases. The cases, they stated, were chiefly interesting by reason of the fact that they illustrated very forcibly how bacteriological examinations might assist the surgeon in his work. The first case was one of kidney infection and one in which no positive diagnosis had ever been made until after the bacteriological examination of the urine, although several diagnosticians had suggested a number of probable causes that included everything from cancer of the kidney to tuberculosis. Briefly the clinical picture was one in which the patient was suffering from a mental disturbance, involving the loss of memory, at times so profound as to approach loss of reason. This was not a constant condition but was periodic, and it was

noticed that the period of greatest disturbance occurred at the time that the urine of the patient was clear. This led to the bacteriological examination of the urine at a time when the patient was improved and the urine was very cloudy. The results of this bacteriological examination were as follows: The hanging drop showed many quiescent leucocytes and an immense number of non-motile bacteria; these latter were principally in the form of short bacilli with rounded ends, singly and in twos, with occasional short threads and coccus forms. Poured and streaked agar plates showed only two varieties, one greatly in the minority, from which was isolated the *bacillus fluorescens liquefaciens* (probably a contamination). The large majority of the colonies were composed of an organism having the following characteristics (in twenty-four-hour cultures): a short, thick, non-motile pleomorphic bacillus with rounded ends, occurring singly and in twos, with occasional short threads and coccus forms; it was decolorized by Gram; in glucose broth at twenty-four hours there was marked polar staining; no spores were seen; in twenty-four hours there was abundant production of gas, and milk was firmly coagulated; gelatin was non-liquefied; there was growth along the entire stab, the surface growth was somewhat raised. On surface agar there was an abundant porcelain-like growth. The individual colonies had entire margins. On potato there was a yellowish-white abundant growth, with formation of gas. Three cubic centimetres of a twenty-four-hour broth culture inoculated into a guinea-pig intraperitoneally caused death in twenty-four hours. It would be seen that the characteristics of this organism corresponded very closely with those of the *B. aërogenes* as given by Flügge. It must be placed, therefore, in the *coli communis* group as a variety of that organism. The mental condition of the patient was now accounted for

by toxæmia resulting from the poisons produced by the activity of the organisms retained in the pelvis of the kidney by occlusion of the ureter. When there was no communication between the bladder and the kidney, due to this occlusion, the urine would become clear and the mental symptoms become progressively worse until the flow of urine was again established, with consequent free and natural drainage into the bladder. After this natural drainage the mental symptoms would clear up until there was another stoppage of the ureter. This occlusion of the ureter, with the consequent symptoms, was occurring with such frequency that the advisability of draining the kidney by operation was considered. Here the surgeon was confronted by a condition that might well make him shrink from advising such an undertaking; in fact, all of the consultants did advise or suggest the expectant plan which in this instance simply meant waiting for the patient to die. These were the conditions: the patient, a lady, seventy-three years old, was suffering from frequently recurring attacks of toxæmia, and as a result thereof was in a weakened and debilitated condition which made the prognosis a matter of days only (without the added shock of operation), and the possibility that after all the operation might not relieve the symptoms. It was no wonder that the surgeons hesitated. The operation was performed, the kidney drained from the outside, and the patient made a splendid and complete recovery. A few weeks after the operation they made cultures from the open sinus and the urine from the bladder as well, with a view to determine what organisms were present; the surgeon undoubtedly having in mind the advisability of allowing the sinus to close. The cultures were interesting from the fact that they showed the same organisms present in the urine and in the lower end of the sinus, which communicated with the pelvis of the kidney, these



being identical with the principal organism found in the previous examinations. The outer end of the sinus and the pus from the drainage tubes showed mixed infection in which the staphylococcus pyogenes aureus was predominant. The second case was that of a lady who suffered with considerable pain in the loins, had a slight elevation of temperature, and whose urine was somewhat cloudy and contained pus cells. The urine was collected under favorable conditions and sent to the laboratory for bacteriological examination; there it was found to contain several million bacteria for every cubic centimetre. In the poured agar plates two varieties were found in about equal numbers. One resembled that found in the first case, except that it possessed a moderate degree of motility; the other showed less abundant growth on all the culture media, the organisms were longer, more slender, showed polar staining, and were non-motile. In all other characteristics the latter resembled the first culture. They must, therefore, be classed as varieties of the bacillus coli communis. The diagnosis of infection of the pelvis of the kidney was made and operation arranged for. It was suggested that before operation the ureters be catheterized and the urine from them examined; when this was done it was found that the urine directly from the ureters was practically free from organisms, while that from the bladder showed the same number as before. From these examinations it was evident that the main infection was of the bladder. Operation on the kidney was given up and the bladder washed out with weak boric-acid-formalin solution, and the patient made a good recovery.

#### *Discussion.*

Dr. W. H. PARK said that he had been much interested in these two cases on account of the sharp contrast be-

tween them. In the former case the surgeon had been loath to operate until encouraged by the results of the bacteriological examination; in the second the surgeon had been on the eve of operating on the kidney, when restrained by the bacteriological examination. The practical value of such examination was well emphasized in these cases.

#### REMARKS ON SOME USES OF PARAFFIN IN BACTERIOLOGICAL WORK.

Dr. PARK spoke on this subject. In the growth of tetanus and other anaërobic bacteria a layer of paraffin melting at 42° C. on top of the culture media had been found to give very good results. The growth of the bacteria and the development of toxins had been as rapid as under the usual methods. The very hot sterile melted paraffin could be poured on after the inoculation of the media, or inoculation could be made through the fluid paraffin, the culture media and paraffin having been sterilized together. The method seemed to be simple and effective. The paraffin could be readily separated from the glass by gentle pressure and the culture drawn off if desired.

#### A FURTHER REPORT ON THE ACTION OF MODERATE AND INTENSE COLD ON BACTERIA.

Dr. PARK made this report. He said that the original report had been on the effect of cold on the typhoid bacilli in water. Twenty cultures had been frozen for varying lengths of time. On an average the cultures showed 42 per cent. of the bacilli living for half a week; 14 per cent. for one week; 7.5 per cent. for two weeks; 0.4 per cent. for three weeks; 0.1 per cent. for five weeks; .09 per cent. for seven weeks; .05 per cent. for nine weeks;

.005 per cent. for twelve weeks; .002 per cent for fifteen weeks; .0001 per cent. for eighteen weeks. At twenty-two weeks all of the bacilli in the twenty cultures were dead in the water. For the first three weeks all of the cultures were alive in the water. There was evidently quite a difference in the viability of the cultures. Experiments had also been made with the bacteria exposed to liquid air for different periods. At the end of three minutes only 18 per cent. of the typhoid bacilli were living; at the end of sixty minutes only 7 per cent. The colon bacillus acted very similarly. The hay bacillus with spores showed 55 per cent. living at the end of two hours. The streptococcus and diphtheria bacillus were tested for virulence after two hours, and this was found to be unaltered. Motile bacteria were found to be but little excluded from water in the act of freezing.

#### TUBAL ABORTION.

Dr. H. J. BOLDT presented a specimen showing very well the process of tubal abortion. At the uterine extremity the tube was entirely occluded, while the abnormal extremity was well open. As a rule, the embryo was expelled in the course of a few days, but from the history in this case the process of expulsion had been going on for about three weeks. It was not common to have much hemorrhage from the vaginal route, yet in this case the hemorrhage had been so profuse that the clots were as large as an ordinary intrauterine abortion.

#### SPONTANEOUS DIASTASIS OF THE CAPUT FEMORIS.

Dr. V. P. GIBNEY presented several specimens. The first one was of spontaneous diastasis of the caput femoris. Two days ago he had proceeded to excise the head of the femur in a child having a mid-dorsal Pott's disease and dis-



ease of the hip joint. The presence of a fluctuating tumor above the trochanter major had led him to attempt excision. On making the incision a little pus had been evacuated, and his finger had then felt the head lying loose in the acetabulum, and as sharply separated from the acetabulum as if it had been done with a saw or chisel. The acetabulum was perfectly smooth, but within the head of the bone was softened tissue which the pathologist thought to be tuberculous. He had seen only one other similar case, and in that one the head had been found lying in Scarpa's space.

#### OSTEOMYELITIS OF THE FEMUR.

The second specimen was one of osteomyelitis of the femur. The child had developed the osteomyelitis about one year ago. Three months after admission it had been found that the left knee was apparently the seat of a phlegmonous inflammation. Two weeks later thick pus had been removed by aspiration. Finally an incision had been made on either side of the knee, and the pus evacuated. Two weeks later the incision had been enlarged. The tissue was found to be tuberculous. Since that time there had been no attempt at healing, and consequently a few days ago the thigh had been amputated. The centre of the bone had been found broken down and fetid. On sawing through the bone the epiphysis was found to have disappeared, and only the shell of bone remained. The pathologist had been unable to arrive at any conclusion regarding the etiology of the osteomyelitis or of the tuberculous process.



“Middleton-Goldsmith” Lecture

---

ON THE ETIOLOGY OF TROPICAL  
DYSENTERY

By

SIMON FLEXNER, M.D.,

Professor of Pathology, University of Pennsylvania.





## ON THE ETIOLOGY OF TROPICAL DYSENTERY<sup>1</sup>

By SIMON FLEXNER, M.D.

There are few subjects in medicine that have attracted more attention than dysentery. Its history dates from the earliest written records and its ravages, unlike those of many of the pestilential diseases, have continued practically unaltered to the present day. The most ancient writing upon medicine—the papyrus Ebers—contains allusions to dysentery; the oldest Indian medical writers refer to it under the name “Atisar,” while Herodotus speaks of its prevalence in Thessaly. Hippocrates, however, was the first to regard dysentery as an independent disease. I should hesitate to bring before this audience a subject so time-worn were it not for the fact that the nature and more especially the etiology of dysentery are among the problems that still await a satisfactory solution. Although the destructive epidemics which characterized the appearance of the disease in ancient times, and which were not unknown even as late as the last century, are in our own day encountered only as accompaniments of war and famine; nevertheless, dysentery still occurs in epidemic form in many Eastern and Western countries, while in the tropics the disease—not unlike cholera, another infectious disorder, the characteristic lesions of which are situated in the intestines—would seem to have found an endemic home.

<sup>1</sup> “Middleton-Goldsmith” Lecture. Delivered before the New York Pathological Society, April 12, 1900.

Our imperfect knowledge of the nature of dysentery should be ascribed neither to lack of opportunity for the study of the disease nor to lack of energy in its pursuit. The literature contains some of the most distinguished names among clinicians and investigators, thanks to whose efforts its clinical history, its epidemiology, and, to a less extent, its pathological anatomy have received partial elucidation. Nor has the disease, in the past quarter of a century, escaped the attention of the bacteriologists, although it must be confessed that the results of somewhat extensive studies along these lines have been far less conclusive than might have been expected. Given a disease that is never entirely absent from temperate and tropical regions, that appears with epidemic severity, that permits of easy access to the *materies morbi*, one would certainly have been tempted to predict that the success achieved in so many other and apparently no less difficult fields would probably be repeated. That the attempt to establish a common etiological factor for all cases of dysentery has thus far failed, this audience need not be reminded. That this failure has tended to emphasize the existence of several pathological states, for which the term dysentery is employed merely as the collective designation, need not be maintained here. But that these conclusions regarding the disease may after all not be in keeping with the facts is at least open to suspicion. When we recall the protean nature of other infectious diseases, among the most common of which are tuberculosis and typhoid fever, there can be no *a priori* objection to the hypothesis that the causative agent of dysentery need not necessarily vary for each of the many types of the disease that have, from time to time, been distinguished.

For the purpose of my inquiry, I shall consider briefly the clinical and pathological types, after which I shall ask your attention to the evidence for belief in specific causes.



In considering this topic I shall endeavor to bring out the bearing of such studies upon dysentery in general and particular types of the disease. Finally, I shall hope to emphasize certain considerations connected with the etiology and pathology of dysentery by reciting some observations made upon the dysenteries prevailing in the Phillippine Islands.

*Types of Dysentery.*—That the lines of demarcation between the several clinical and pathological types should be inaccurate is not a matter of wonder. Both the beginning and the end of any given instance may vary widely, and the symptoms and lesions of cases arising sporadically in temperate climates may agree with those of dysentery occurring endemically in the tropics, or epidemically in both localities. The terms "catarrhal," "tropical," "epidemic," and "diphtheritic," are far from signifying sharply-defined entities. The clinical manifestations and pathological lesions of the catarrhal variety occur in all kinds of dysentery and in all places where the disease prevails. Ever since the time of John Hunter there have been those who, upon pathological-anatomical grounds, have separated the endemic from the epidemic disease, and the line has been even more sharply drawn during the past decade, since the discovery of the *Amœba coli* in its relations to tropical dysentery. But the distinction between tropical dysentery and the epidemic disease is far from being sharp and constant. Diphtheritis and ulceration are not safe criteria. For while the former is commonly present in the epidemic disease, it occurs also in the tropical malady, and may, according to Kartulis, be associated with the ulcerative amœbic variety, in which the lesions begin with destruction of the submucous coat of the gut.

As must always occur when classification of a disease proceeds upon clinical and pathological rather than

etiological lines, the literature of dysentery is burdened with an interminable mass of appellations indicating the nature of the disorder or the author's conception of the pathological anatomy. Dysenteries, however, are now divided by the chief writers into several groups, depending upon the clinical history or the mode of prevalence. Thus Osler writes of four varieties, the acute catarrhal, the tropical or amœbic, the diphtheritic, and the chronic dysentery. Davidson considers the subject under two headings: (1) according to prevalence—epidemic, endemic, the dysentery of war and famine; (2) upon clinical grounds—acute fibrinous or pseudo-diphtheritic, and chronic dysentery. Kartulis describes endemic, epidemic, and sporadic varieties; Manson speaks of a catarrhal and ulcerating dysentery, while Delafield distinguishes in the environs of New York at least five distinct types of this disease, only one of which would appear to be due to a specific agent—the *Amœba coli*.

*Evidences for Specific Causes. Bacteria.*—The presence of bacteria in the stools and tissues in dysentery was demonstrated by Klebs, Prior, and Ziegler, whose studies, carried out upon the epidemic disease, have now only a minor historical interest, although Ziegler still holds that the relations of certain bacilli to the lesions speak for their pathogenic action. The early studies of Hlava upon the epidemic disease yielded quite inconclusive results, since, although he was able to obtain as many as nineteen different kinds of bacilli in cultures, inoculations into animals failed entirely to reproduce the morbid process. Chantemesse and Widal were somewhat more fortunate. From five cases of tropical dysentery they obtained a bacillus which, when injected into the stomach or rectum of guinea-pigs, gave rise to diphtheritis, an observation which Grigoriew, who believed that he had isolated the same micro-organism from ten cases of dysentery, failed to confirm.



Maggiori, who studied eleven cases of the epidemic disease, obtained *B. coli communis* regularly and in large numbers. Less frequently *B. proteus vulgaris* was isolated, while in some cases the pyogenic cocci and *B. pyocyaneus* were found. This investigator considered it highly probable that the disease was caused by a *Bacillus coli* of intensified virulence—a conception also shared by Laveran, Arnaud, Celli and Fiocca, and Escherich, who isolated the same organs from dysenteric cases. Arnaud's series was larger, embracing fifty-three acute cases occurring in Tunis, from all of which *B. coli* was isolated. The spleen of a fatal case also yielded him the organisms. The ingestion of cultures in the case of several kinds of animals gave no results; while the rectal injections of bouillon cultures, previously raised to 60-80° C., produced in two dogs a fatal and characteristic dysentery—a result in striking opposition to those reported by other writers.

The studies upon this bacillus by Celli and Fiocca are the most important which we possess. Their cases numbered sixty-two, and included examples of the sporadic, epidemic, and tropical disease occurring in Italy and Egypt. From the fact that especial attention was paid to the occurrence and action of the *Amœba coli*, the results of these authors are doubly useful. They exclude this organism as the cause of any form of the disease and consider that a variety of the colon bacillus, of especial virulence, which they designate *B. coli dysenteriae*, is responsible for the lesions. Along with this colon bacillus they found typhoid-like bacilli and streptococci. Their experiments upon animals are also more conclusive than any others. With cultures they were able to produce dysentery in cats, and while they admit that other micro-organisms were also capable of producing similar results, they found that the latter acted far less constantly than their dysenteric



bacillus. A toxin separated from growths of the organism was found to give rise to similar conditions. Celli, in another publication, expresses the view that it is this toxin that first exerts an injurious effect upon the intestinal mucosa, after which the destructive lesion is produced by the pyogenic cocci. Results similar to those of Celli were obtained by Del Pino and Alessandri. The latter, working on a case of post-operative dysentery, secured cultures of *B. coli* that yielded a toxin capable of setting up dysentery in young cats. In their most recent paper, Celli and Valenti describe the production in dogs of sera, which, when tested upon experimental animals, exert a protective and healing effect. Upon human beings its action was not equally positive.

The colon bacillus is also believed by Escherich to play an important part in the production of a contagious enteric disorder (*colitis contagiosa*) in children. In its morbid anatomy the disease agrees with catarrhal dysentery.

The bacilli thus far enumerated, except those of Chantmesse and Widal, so far as they could be studied in cultures, have shown no specific properties. They all represent a well-known bacterial species, constantly present normally in the situation from which the organisms were obtained in disease, and whose only unusual properties were increased virulence when tested upon animals, and a capacity to set up enteritis when injected into the intestines of dogs and cats.

The investigations of an epidemic of dysentery, which prevailed in Japan, yielded different and apparently more convincing results. Ogata isolated fine bacilli, which liquefy gelatin, stain by Gram's method, and set up, when introduced by the mouth or by the rectum into guinea-pigs and cats, intestinal ulcerations. The organism regarded by Ogata as the cause of epidemic dysentery was isolated from twenty-three cases of the disease occurring

in Padua by Vivaldi. Since that time it appears not to have been found again.

This list does not entirely cover the bacillary species isolated from cases of dysentery. The recent publication of Shiga, who also studied the disease prevailing in Japan, is needed to complete the number. But as this investigator's studies have a very direct bearing upon my own, I shall defer speaking of them for the present.

On the other hand, a causative rôle in the production of dysentery has also been ascribed to the pyogenic cocci. Besides being found in association with bacilli, by several of the investigators already mentioned, they have been regarded as the chief pathogenic agents by Zancarol, whose studies were carried out in Alexandria, by Silvestri of Turin, by Bertrand and Baucher of France, and very recently by Ascher, who investigated cases arising in Eastern and Western Prussia. These cocci, especially the streptococci, were capable in certain instances (Silvestri, Ascher) of setting up, in cats, dysentery, and liver-abscess. The cocci isolated by Ascher were also said to have shown the agglutination reaction with the blood-serum of the patients from whom they were obtained. As a cause of a special variety of endemic dysentery—the endemic enterocolitis of Cochin China—Calmette obtained the *Bacillus pyocyaneus*. The same micro-organism was isolated, from a small epidemic of the disease occurring in New York State, by Lartigau, in another epidemic in children, prevailing in Canada, by Adami, and in certain sporadic cases of gastric and enteric infection, by L. F. Barker in Baltimore.

*Protozoa*.—Because of the great diversity of the normal intestinal flora the varieties of bacteria which can be isolated from the dejections and intestinal contents are relatively numerous. The differences in the numbers and kinds of bacteria capable of flourishing there, rendered



possible by the existence of pathological conditions, readily account for many of the results of the bacteriologic studies given. With animal microparasites the case is different. Protozoa do not exist in easily demonstrable forms or numbers in the dejecta in health, and the number of species occurring under all conditions is small. Of these the list is confined to a few kinds of flagellates—which even when present in considerable numbers do little harm—and the amœbæ.

It is to the rôle of the amœbæ—a particular variety of which has achieved the distinction of being connected in a causal relation with endemic dysentery—that I wish to direct your attention. Since the studies of Kartulis, Councilman and Lafleur, and Kruse and Pasquale, so firmly has the idea of this connection taken hold of the popular medical mind, that the designation “amœbic” as synonymous with “endemic” or “tropical” dysentery has been widely adopted. And yet the evidence upon which this belief is based cannot be regarded as convincing. Until we shall have gained means of differentiating amœbæ other than those we now possess, and, moreover, until we are able to control their development with at least as great perfection as in the case of bacteria, the question of the precise part played by them in dysentery cannot be satisfactorily determined.

Since the historical observation of Lambl upon the occurrence of amœbæ in the dejections of human beings, and especially since the confirmatory observations of Cunningham, Lewis and R. Koch, our knowledge of the distribution of these organisms in human beings has constantly widened and deepened. It was, moreover, the study by Löscher of a case of dysentery in which an amœba, called by him *Amœba coli*, occurred that gave the impulse to the investigation of the pathological actions of such organisms.



The importance of amœbæ was further enforced by the demonstration of the organisms in sections of dysenteric ulcers by R. Koch, first in Egypt and afterwards in India. Following these successes, and probably directly through the suggestion of Koch, Kartulis began his series of observations and publications, which more than any other similar writings have tended to isolate tropical dysentery and place it upon a probable etiological basis.

The work of Kartulis need not be reviewed in detail in this place. His conclusion that in every undoubted case of dysentery occurring in Egypt the amœbæ are present, has not been entirely confirmed. That they are, moreover, never present in the intestines in other enteric diseases and in health has also not been the experience of all other investigators. But that amœbæ are abundant in many cases of tropical dysentery and may also be demonstrated in the contents of the hepatic abscess in the disease, the studies of Kartulis and those of subsequent investigators have definitely shown.

To follow the list of discoveries in cases of dysentery which have accumulated in the last decade is unnecessary and would carry us too far afield. Reports confirmatory of the results of Koch and Kartulis have appeared from many European countries, the Pacific Islands, and from both North and South America. Ten years have, however, seen a modification of the views regarding amœbæ, as causes of pathological conditions in human beings. The demonstration of amœbæ, indistinguishable from the *Amœba coli*, in various intestinal diseases and even in healthy persons, has necessitated a recasting of the exclusive belief in their pathogenicity and relation to dysentery.

That amœbæ may exist in diseases other than dysentery was conclusively proven by early observations (Cunningham, Lewis) upon choleraic discharges. More important results were obtained by Grassi, first in 1882 and later in

1888. He describes amœbæ which may occur, in considerable numbers, in diseases as varied as typhoid fever, cholera, pellagra, and colitis secondary to tumors. He demonstrated their presence in diarrhœas and dysenteries, but at times also found them in the dejecta of healthy individuals. That amœbæ can exist in the intestines without any disturbance of the health of their host was, therefore, definitely established by Grassi's observations as well as by those of Calandrucci, Massiutin, Kruse and Pasquale, Gasser, and Schuberg. The observations of the last-named investigator are especially valuable, in that he showed that to the reaction of the lower colon and the consistence of the fæces was due the fact that amœbæ could not be constantly found with comparative ease in the dejecta. If a laxative, as for example, Carlsbad salts, is administered and the contents of the upper colon are then collected and examined, amœbæ are frequently demonstrable. Moreover, under these conditions flagellates—the trichomonas and cercomonas—appear in the stools. He looks upon these parasites as common commensals in man.

It can, therefore, no longer be held that amœbæ are necessarily pathogenic when found sojourning in the intestine in man. Indeed, the necessity of recognizing distinct species of amœbæ had already begun to force itself upon the thoughtful students, who until lately believed in their constant pathogenic action. Quincke and Roos, who observed amœbæ first in a healthy individual, and later in two widely dissimilar cases of enteritis, tested the different organisms concerned for pathogenesis upon cats. From their results they believed that according to certain structural differences, and the action of the parasites in engulfing blood-corpuscles, two main varieties or species can be distinguished, the one phagocytic for red blood corpuscles and pathogenic for man and cats—this species being identical with the *Amœba coli* of Löscher—the other,



nonpathogenic and nonphagocytic for blood corpuscles, whi hcthey designate *A. coli mitis*. According to this view *A. coli*, *A. felis*, and *A. dysenteriae* of Councilman and Lafleur are all one species; *A. coli mitis* a harmless saprophyte, or at least is one of relative pathogenic insignificance.

It requires no elaborate argument to bring out the fallacies of such a method of distinction. Results following the introduction of so complex a material as dejecta into an animal cannot be accepted as deciding the properties of any single constituent. What has already been said concerning the ability of pure bacterial cultures to produce enteric lesions affords a sufficient criticism of such a procedure.

That amoebæ when combined with bacteria may cause intestinal lesions and even ulceration is now established. Furthermore, the experiments of Kartulis and Kruse, and Pasquale with the contents of hepatic abscesses, supposed to be free from bacteria, are all but convincing, in so far as they may be supposed to prove the capacity of amoebæ alone to set up such changes.

The attempt to cultivate the *Amœba coli* without admixture of bacteria has in all probability never yet been successfully carried out. The supposed positive experiment of Kartulis is now known to have been erroneous. Whether Celli and Fiocca have succeeded is still doubtful. In any case no satisfactory experimental production of amoebic dysentery in cats or other animals has thus far been achieved with cultures of amoebæ free or relatively free from bacteria.

The pathological findings in amoebic dysentery have been adduced as proving its specific character by Councilman and Lafleur, Kruse and Pasquale, and Kartulis. According to this view the intestinal ulcers in amoebic dysentery begin as in infiltration of the submucous coat



that leads to necrosis of the overlying membrane. The lesions, unless complicated by the presence of bacteria, are free from the products of purulent inflammation. The accompanying abscess of the liver exists independently of the presence of bacteria. Krause and Pasquale, and Kartulis ascribe greater significance to bacterial association than do Councilman and Lafleur. Kartulis sees in the occasional diphtheritis evidence of their action, while Kruse and Pasquale have followed them in their penetration into the coats of the gut, where they lie side by side with the amœbæ or even precede them in the invasion. Both bacilli and cocci occur; and wherever necrosis is found, bacilli are sure to occur in groups and masses.

"The amœbæ and bacilli together start the lesions in the intestine." (Kruse and Pasquale.)

The form of lesions here described does not entirely represent the disease as it appears in the tropics. Even Lafleur in a later communication says that the term "tropical" can be used only as a partial synonym for amœbic dysentery, inasmuch as, on the other hand, the disease, though more frequent in the tropics, is by no means limited to those regions, while, on the other hand, there are undoubted cases of dysentery in the tropics which are not of the amœbic form—a statement borne out by my own observations.

How far, then, have the foregoing results aided us in clearing up the hitherto obscure etiology of this protean malady? While epidemic, endemic, and sporadic dysentery have been subjected to bacteriological investigation, the chief work thus far done has been with the tropical variety. Although the actual number of cases of the epidemic disease which have been carefully studied is small, it is significant that, with two exceptions—the studies of Ogata and Calmette—the several different micro-organisms isolated have all been bacteria which are

normally present in the intestinal canal. The cultures have, it is true, undergone changes of virulence, but in other respects they have retained their ordinary biological and physiological properties unaltered. It would certainly seem *a priori* highly improbable that so severe and devastating a disease as epidemic dysentery should be due to micro-organisms which are constant inhabitants of the intestinal tracts. Moreover, when it is recalled how easily and under what great variety of conditions such accessions of virulence may be achieved, it would be remarkable that epidemics of dysentery are, nevertheless, relatively rare phenomena.

Turning to the tropical disease, we also observe that several micro-organisms have been assigned as the causative agent. Chantemesse and Widal have described a bacillus not obtained since from similar cases; Arnaud, Laveran, and Celli and Fiocca have seen in the colon bacillus, modified in its pathogenicity, a sufficient cause. More weight, and with far greater justice, has been laid upon the *Amœba coli*. But this organism has been found more especially in chronic cases. Kartulis states that the catarrhal stage, which is common in the epidemic and sporadic disease, is uncommon in this form. If the *Amœba coli* is the organism most commonly present in the acute dysenteries of the tropics, the fact has not yet been conclusively established. However this may be, in Manila, where the organism is not infrequently present in the chronic disease, its absence in the very acute and often rapidly fatal cases of dysentery must be regarded as of significance.

From the preceding considerations the following conclusions seem warranted:

1. No bacterial species yet described as the cause of dysentery has any especial claim to be regarded as the chief micro-organism concerned in the causation of the disease.

2. It is unlikely that any bacterial species that is constantly and normally present in the intestine or in the environs of man, except where the disease prevails in an endemic form, can be regarded as the probable cause of epidemic dysentery.

3. The relations of sporadic to epidemic dysentery are so remote that it is improbable that the two diseases are produced by the same organic cause.

4. The pathogenic action of the *Amœba coli* in many cases of tropical, and in certain examples of sporadic, dysentery, has not been disproved by the discovery of amœbæ in the normal intestine and in diseases other than dysentery. While amœbæ are commonly present and are concerned in the production of the lesions in subacute and chronic dysentery, they have not thus far been shown to be equally connected with the acute dysenteries even in the tropics. In the former varieties, bacterial association probably has much influence upon the pathogenic powers of the amœbæ.

#### THE DYSENTERY OF JAPAN AND THE PHILIPPINE ISLANDS.

Every year, especially in the summer and autumn, dysentery prevails in Japan. Ogata and Eldridge have given the statistics of incidence and mortality in the years from 1878 to 1899:

Year.	Cases.	Deaths.
1878.....	1,118	206
1879.....	8,322	1,487
1880.....	5,047	1,305
1881.....	7,001	1,837
1882.....	4,330	1,313
1883.....	21,172	5,066
1884.....	22,702	6,036
1885.....	47,377	10,690
1886.....	24,328	6,839
1887.....	16,125	4,244
1888.....	26,789	6,570
1889.....	22,893	5,970



Year.	Cases.	Deaths.
1890.....	42,632	8,706
1891.....	46,358	11,208
1892.....	70,842	16,844
1893.....	167,305	41,282
1894.....	155,140	38,094
1895.....	52,711	12,059
1896.....	85,876	22,356
1897.....	91,077	23,189
1898.....	90,933	22,379
1899.....	125,989	26,709
	1,136,067	275,289

The epidemic studied bacteriologically by Ogata occurred in the province of Oita where in 1890, 801 cases occurred with 221 deaths, and in 1891, 8390 cases with 2163 deaths—an average mortality of from 26 to 27 per cent. This epidemic had been preceded by sporadic cases in the previous winter, and in its spread showed a striking contagious character. The lesions in the intestines are described in one case, death having taken place on the eleventh day of the disease. The lower segment of the small intestine was hyperæmic. The large intestine was greatly swollen so that the lumen was almost obliterated. The mucous membrane was hyperæmic and presented a deep bluish-red color. The contents were chocolate colored. The mucosa of the transverse and descending colon, and especially of the sigmoid flexure, showed small ulcers, the size of peas, which were so numerous as to give to the membrane a sieve-like appearance. They were also found, though in smaller numbers, in the descending colon and rectum. No large ulcers were present. The peculiar bacilli—staining by Gram, liquefying gelatine, and causing in animals, whether injected beneath the skin or into the rectum, hyperæmia and ulceration of the intestinal mucosa—have been already described. Ogata's decision was that the bacillus isolated was probably the cause of the epidemic of dysentery prevailing in Southern Japan.

I am not acquainted with a full description of the morbid anatomy of the dysentery prevailing in Japan. Schaube formerly of Tokio, in his *Die Krankheiten der warmen Länder*, does not give a detailed account of the Japanese variety, but contents himself with the usual classification and description of the disease. The meager accounts may possibly be explained by the fact that necropsies are obtained in Japan only with great difficulty. Dr. Eldridge states that the lesions seen in "amœbic dysentery" as described by Councilman and Lafleur are seldom met with. The common lesions are destructive, progressing from the surface downwards, associated with the necrosis of the mucosa and croupous infiltration (diphtheritis). Perforation is unusual, the muscular coat offering a strong resistance to the pathological process.

The most recent bacteriological study of dysentery has been made by Shiga, to whose results I would ask especial attention. During 1897 the disease prevailed epidemically in Japan, 89,400 cases with 22,300 deaths (24 per cent.) being recorded from June to December. Out of a considerable number of cases occurring in Tokio, 36 were subjected to bacteriological examination by Shiga.

As was readily recognized by him, four points must be proved in the effort to show that an organism suspected of standing in etiological relation to any given disease is really the causative agent: (1) The organism must occur constantly; (2) it must be a species not present normally in the diseased part; (3) it must be pathogenic and produce in experimental animals lesions similar to those from which it was obtained; (4) it should in virtue of its pathogenic activity in man show the Widal agglutination reaction with the blood-sera of those who have suffered from the disease. From the series of cases examined there was obtained from the dejecta and intestinal contents and walls, and from the mesenteric glands, a bacillus which

fulfilled all these requirements and which was regarded as the cause of Japanese dysentery, at least.

Before entering upon a description of this organism, I should like to direct your attention to the dysentery prevailing in the Philippine Islands, especially in and around Manila. The report of the Surgeon-General of the Army for 1899 contains a tabulation of diseases, observed among the American troops, during the first four months of the American occupation of Manila. In it the dysenteries are included with the diarrrhœal diseases. The total number of cases reported is 445, the death-rate being 0.48 per cent. The comment made is that "the malarial diseases exceed their prevalence in the United States in the proportion of 370 to 96, and the diarrrhœal diseases in the proportion of 445 to 116, or about 4 to 1 in both instances."

This compilation fails to give an adequate idea of the extent, severity, and mortality of dysentery in Manila. Although, unfortunately, figures are not obtainable, I am convinced, after nearly three months' residence in Manila, that the enteric diseases, of which dysentery was the most frequent and important, were the chief causes of disability and mortality among the land forces of the American army.<sup>1</sup>

The disease appears in two main forms, acute and chronic dysentery. The stools and intestinal contents at autopsy were scrutinized for amœbæ. So far as regards the acute cases, these organisms were absent or very difficult to find in the fresh stools and in the intestinal contents immediately after death. In the chronic forms of the disease ulcers were present in the mucosa and submucosa;

<sup>1</sup> The studies on dysentery here recorded were made by a commission consisting of Dr. L. F. Barker and myself, sent out by the Johns Hopkins University to study the diseases prevailing in the Philippine Islands. To this commission were attached Messrs. Joseph M. Flint and Frederick P. Gay, who were, at that time, members of the Johns Hopkins Medical School.



the lesions were confined to the large intestine, the coats of which were greatly thickened; at times large sloughs of the mucous membrane, partly detached, were encountered. Amœbæ were commonly present, but were variable as to actual occurrence and numbers. Large hepatic abscesses, usually single, were encountered in a number of these cases. Amœbæ were not always found in the contents of these abscesses; sometimes bacteria were present alone or associated with amœbæ. Amœbæ not distinguishable, except by the absence of specific inclusions, from those in the stools of human cases exist in the dejecta of monkeys liberated from captivity and now at large in Manila.

The morbid anatomy of the chronic disease agrees in part only with that of "amœbic dysentery." I shall draw attention later to another form of the chronic disease.

The pathological changes in the acute cases differ widely from those of the chronic affections. I shall now give in brief the condition of the intestines in several cases of acute dysentery. The patients were American soldiers.

CASE I.—Death on the sixth day of the disease. The entire large intestine from the cecum of the rectum is dilated and the walls of the gut are thickened. The mucous membrane is swollen, its consistence is much increased and the normal folds are thrown into elevated coarse corrugations. The general color of the mucous membrane is deep red, but there are present many brighter spots evidently due to hemorrhage. No distinct false membrane is to be made out, but here and there are scattered white elevations, which, after the intestine has been washed, become more prominent and can be removed only with some difficulty, small defects in the membrane being left behind. In the fresh state ulceration was not made out, but after the washing referred to, there are found in the lower portions of the sigmoid flex-

ure minute, sieve-like points, with perfectly sharp edges, representing defects which lead into the submucosa. The smallest of these openings are the size of pin-points, the largest about 2 mm. in diameter. The contents of the intestine showed no amœbæ.

CASE II.—Death in the fifth day of illness. The small intestine, excepting the lower end of the ileum, which is deeply congested and swollen, shows no alteration. The serosa over the large intestine is injected, but is otherwise normal. The large intestine is much thickened and its consistence is increased. On opening the gut the contents are found to be dark or nearly black in color, an appearance probably due to the administration of bismuth. The mucous membrane extending from the rectum to the ileocecal valve, and beyond the valve in the ileum for a distance of 4 cm., is congested, swollen, and hemorrhagic. Scattered here and there on the surface are elevated, white, irregular points and small, flattened areas, which suggest a pseudo-membrane, but which cannot be absolutely identified as of such a nature. The normal velvety character of the mucous membrane is lost. No ulceration can be made out and the lesions seem to be chiefly in the mucosa and submucosa. Particularly conspicuous is the wide diffusion of the lesions, no part of the mucous surface within the limits defined having escaped. The mesenteric glands are congested and moderately swollen. Although amœbæ had been found in the evacuations two days previous to death, they could not be demonstrated in the intestinal contents at autopsy.

CASE III.—Death after an illness of six days. The large intestine is markedly dilated and the serous coat is much injected. The contents of the large intestine are represented by a grumous, pink, pulpy material. The mucous membrane is swollen and hyperæmic, and represents a striking granular appearance due to exudate upon

the surface; many areas of hemorrhage are also observed. The entire mucosa of the large intestine is implicated in this process. Ulceration is not present. The mesenteric glands are swollen, congested, and hemorrhagic. The spleen is moderately enlarged. Amœbæ were not demonstrable in the intestinal contents.

The three cases which have been selected do not agree with amœbic dysentery as hitherto described. In only one were amœbæ found in the stools, and even then they could not be demonstrated in the intestinal contents, obtained immediately after death, which took place two days after the first examination. The stools consisted, as was the rule in the acute disease observed in Manila, of mucus and blood. The microscopical examination revealed epithelial cells, red blood corpuscles, a moderate number of bacteria and many amœbæ. On the same day injections of quinine were begun, ipecac being administered by the mouth. The note states that twenty-four hours later the stool contained mucus but less blood. Epithelial cells were still present and the bacteria appeared in greatly increased numbers. No amœbæ could be discovered. Death took place on the day of this examination.

In their pathological histology also the acute dysenteries differ from the amœbic form. The histological changes appear in the mucous membrane, submucosa and muscularis, being most marked in the former situations. Those of the mucous membrane consist of coagulative necrosis with exudation of fibrin and polymorphonuclear cells. The fibrinous and cellular exudate may entirely replace the glandular layer, or here and there a gland may be preserved. The pseudo-membrane is a close-meshed network of fibrin enclosing multinuclear, often fragmented, cells. No blood-vessels are to be distinguished, but a variable number of red blood corpuscles are mingled with the exudate and lie free upon the surface. The muscu-



laris mucosa is not always distinguishable—indeed it is frequently lost in the exudate. The submucosa is always much altered. From the changes found in it, it is evident that to them is chiefly due the thickening of the gut. The part most affected is the layer next the muscularis mucosa. Here are found hemorrhages of variable size, while in the interstices of the tissues some fibrin appears. More marked, however, are cellular accumulations, which are present, not uniformly, but in irregular areas. The deeper layers of the submucosa show similar cellular infiltrations, although the amount is less striking. On the other hand, at these levels the quantity of fibrin is greatly increased and hemorrhages are numerous.

The character of the cellular exudate is quite uniform. Excluding the red blood corpuscles, the new cells consist chiefly of plasma cells. These are collected into foci, often about blood-vessels, veins and arteries, but sometimes occur in small groups or singly. There can be no doubt that these are identical with Unna's plasma cells; they show the reticulated nucleus, often placed eccentrically, and the fine blue granulations of cell-protoplasm in eosin and methylene-blue staining. As the deeper levels of the submucosa are reached, hemorrhages and fibrin are abundant. The size of the foci of plasma cells gradually diminishes. At the muscular border they have about disappeared. Among the plasma cells a variable number of eosinophilic cells may be distinguished.

In the submucosa, infiltration, hemorrhage, and fibrin-formation take place also beneath an intact or almost intact mucous membrane. The nature of the cellular infiltration may be identical with that already described, but in addition accumulations of lymphoid cells may frequently be seen. These exist in the layer of the submucosa immediately next the muscularis mucosa; the deeper cells resemble plasma cells.

The blood-vessels of the submucosa may be patent and congested, the blood containing an excess of white element or they may show recent leucocytic and fibrinous thrombi. Hyaline degeneration of the vascular walls was not encountered. Large spaces in the submucosa may contain fibrinous clots; these are probably dilated and thrombosed lymphatic vessels.

The muscular coat shows only hemorrhages, which may be of large size, although they are usually smaller than in the submucosa. The peritoneal tunic is usually unaltered.

From this brief description it is evident that the main pathological changes take place in the mucous membrane and submucosa and it is clear also that the two tunics may be affected simultaneously or the submucosa may suffer pathological alterations without involvement of the mucosa. So far as could be ascertained from the material studied, in contra-distinction to the condition observed in "amœbic" dysentery, ulceration did not begin in the submucosa, but any defect which may have occurred resulted from exfoliation of the necrotic mucosa and the attached pseudo-membrane.

It is interesting in this connection, to emphasize the fact that the polymorphonuclear leucocyte plays a very insignificant rôle in the process of infiltration in the submucosa, whereas in the affected mucous membrane it is much in evidence. On the other hand, the blood-vessels of the submucosa contain those cells in increased numbers and the cellular and fibrinous thrombi are rich in them. It would appear, therefore, that these cells do not leave the vessels in the submucosa as readily as those of the mucous membrane. That the blood-vessels of the submucosa suffer great injury from the pathogenic agent is shown by the free hemorrhages and the fibrinous exudation.

Bacteria are abundant in the fibrinous exudation in the

mucous membrane. The chief varieties distinguishable are cocci and bacilli. In specimens stained by Gram's or Weigert's methods, large numbers of cocci, in short chains and groups, can be made out. In other specimens, stained in Unna's alkaline methylene-blue, besides the cocci many bacilli may be seen. These are quite uniform in size; they present the morphological characters of the colon-typhoid group, from which they could not be distinguished in sections of tissue. While the bacteria are so abundant in the necrotic mucous membrane, diligent search failed to exhibit either bacilli or cocci in the infiltration areas of the affected submucosa. The conviction is therefore forced upon one that the lesions in the submucosa are toxic in origin. Amœbæ were not discovered in the sections.

#### THE BACTERIOLOGY OF PHILIPPINE DYSENTERY.

In the study of the bacterial flora of the disease acute and chronic cases were utilized. The methods of procedure varied only slightly in different cases. The acutely ill being in bed, the evacuations were collected in bed-pans, which a short time before had been carefully scalded. The patients who were about the wards were taken to the laboratory, where cultures could be made immediately from the contents of the rectum. The fatal cases were subjected to autopsy immediately after death. The large gut at different intervals was burned through with a hot knife, and cultures were made before disturbing any of the viscera. Plate cultures in agar-agar were employed. The average number of plates made from a single case was twelve. The material was mixed with bouillon, so as to afford the advantage of a relatively large amount for plating. It was frequently obtained from several different portions of the evacuations or from several levels of the



intestines. Only such plates as contained well-separated colonies were utilized. Control microscopical examinations of the evacuations and intestinal contents were made. It may be mentioned that cercomonades and trichomonades were very common in the diarrhoeal stools. They did not appear to be of pathological significance.

From the separated colonies, agar-slant cultures were made. The growths of the pyogenic cocci as well as those of *B. pyocyaneus* were not pursued further. The former was never absent, the latter was rarely present. The bacillary colonies, which occurred with regularity in the acute disease, could be distinguished according to two distinct types. Their properties are as follows:

*Type I.*—Bacillus of the average size of *B. coli communis*. There is variation in length; almost none in thickness. The individuals are usually separate; sometimes they are united in pairs, but only very rarely do they occur as filaments. The ends are slightly rounded. The bacillus shows moderate motility; Gram's stain is negative.

Growth takes place upon all culture media at the room temperature, but better in the thermostat. Gelatin is not liquefied. The colonies resemble those of *B. typhosus*, being more nearly like them when first isolated from the dejecta than after a period of cultivation outside the body. After many months of such saprophytic growth the colonies become thicker, exhibit a moist surface, and are less translucent. The strokes upon agar-slants show a similar alteration. At first the growth extends but little laterally, but later on it becomes 2 to 3 mm. in width, and generally shows distinct indentations at the edges. Upon gelatin the colonies are more delicate; the stab extends along the line of puncture only, spreading very little at the surface of the medium.

On potato, growth takes place along the line of inocu-

lation and spreads beyond. After some days it is a little elevated and of a pale-brown tint. On unfavorable potatoes the growth is slight, moist, and membranous, resembling, except for the greater amounts of moisture, that of *B. typhosus* when typical.

Sugars—glucose, lactose, and saccharose—are not fermented gaseously. In glucose media a moderate acid production takes place.

Bouillon is clouded diffusely and a sediment forms. There is no production of a pellicle.

Litmus-milk assumes, after 24 to 72 hours, a faint lilac tinge. After the lapse of from 6 to 8 days alkali begins to be produced, which increases in amount until the litmus is rendered deep blue in color. No coagulation of the milk ensues.

Indol is not always formed. Even in sugar-free bouillon it may fail to appear, or it may be produced in small quantities only.

Suitable cultures of this organism, when tested for the agglutination reaction with the blood-serum of persons suffering from dysentery—the host of another individual—give, in many cases, a positive result.

The bacillus is pathogenic for the ordinary laboratory animals. It is abundant in the acute cases in which it may be the predominating organism; it becomes more difficult to find as the cases progress towards recovery or chronicity. In the ordinary chronic dysentery of Manila, in which the amoebæ are commonly encountered, it was not found. It can be cultivated from the dejecta during life, and the intestinal contents, mucous membrane, and mesenteric glands in fatal cases.

*Type II.*—Bacilli which are present in all instances. In the acute cases they may not predominate, being less numerous than the members of Type I. In all others it is the predominating bacterium. The properties vary some-

what, but agree well with those of the group *B. coli communis*. The main variations relate to extent and rapidity with which litmus-milk is reddened and coagulated, and the amount of indol produced. The sugars are broken up with the formation of gas. The morphology is also similar to that of *B. coli*; some specimens are motile at the end of 24 hours; in others motility was not demonstrated.

In agglutination tests the results varied according as the blood of the host or of another individual was employed. With that of the host there was frequently a reaction in low dilutions; with that of another person the reaction was rarely and very inconstantly obtained. The pathogenicity was not tested extensively.

The practical operation of separating the several kinds of bacilli which grew in the plates was to inoculate glucose-agar stab-tubes from the different colonies. In those tubes which, after 24 hours at 37° C., showed no gas, the organisms were likely to conform to Type I.

Before proceeding to the assumption that this organism was concerned with the production of the intestinal lesions of dysentery occurring in Manila, it was necessary to establish its absence from the stools of healthy persons and of those suffering from other diseases. Strong presumptive evidence of its being an unusual inhabitant of the intestine of man may be gathered from the facts already known concerning the ordinary intestinal flora. But as such observations would not suffice for a new region and under new conditions, the organism was searched for in other persons who had been in close association with those suffering from dysentery and also in inhabitants of other parts of the Island of Luzon. The organism was not demonstrated in healthy dejecta or in evacuations of persons (native Filipinos) suffering from beri-beri. A further argument in favor of its restricted distribution



is furnished by its absence from cases of chronic dysentery or the marked reduction in the numbers present.

*Pathogenicity.*—The pathogenicity of the bacillus Type I was studied, soon after its isolation, upon mice and monkeys in Manila, and upon various animals in this country with cultures brought from the Philippines.

*Monkeys.*—Subcutaneous inoculation gives rise to a swelling from which the animal suffers no inconvenience and quickly recovers. Monkeys which were given croton oil and, after purging had been established, 10 cc. of a bouillon culture through a stomach-tube, soon recovered from the effects of the purge, and no further results could be noted.

*Mice* are susceptible to subcutaneous and intraperitoneal inoculations. Death takes place in from twenty-four to forty-eight hours,—rarely after several days,—the reaction varying according to the dose and the mode of inoculation. The site of puncture shows edema and, in the case of injections made beneath the skin, a hemorrhagic exudate. Inoculation into the peritoneal cavity gives rise to a variable amount of faintly turbid exudate and small white flakes of leucocytes; the pleura contains an excess of clear fluid, which is often present also in the pericardium; the serous vessels are injected and small hemorrhages may occur, more especially in the subcutaneous tissues. The superficial lymphatic glands are swollen and congested or hemorrhagic; the spleen is enlarged, the kidneys and adrenal glands are congested; the lungs show a marked congestion and the intestines contain an excess of glutinous contents. Coverslips from the peritoneal and pleural exudates show bacilli, often in large umbers, and polymorphonuclear leucocytes. These cells frequently show engulfed bacilli. Cultures prove a general invasion of bacilli with relatively smaller numbers of organisms in the spleen and heart's blood.

*Guinea-pigs* react in much the same way as mice, larger doses being required to produce fatal results, while the bacilli show less tendency to invade the internal organs. Subcutaneous injections cause a local swelling consisting of pus-corpuscles, serum, and blood; the superficial lymph-glands become swollen, and an exudate appears in the peritoneal and more rarely in the pleural cavities. Intraperitoneal inoculations give more characteristic results. Death takes place in from one to six days, depending upon the source and amount of the culture. The inguinal and axillary lymphatic glands are enlarged and reddened; the peritoneal cavity may contain glutinous fluid and floating whitish flakes of pus-corpuscles, or, with little fluid, there may be grayish-white solid exudates of considerable size over the liver, spleen, and intestines. The blood-vessels are injected, the small intestines are filled with a soft glutinous matter, ecchymoses occur in the mucosa of the intestines, and Peyer's patches may be swollen and reddened. If death occurs late the swelling of the Peyer's patches may have disappeared and be represented by the "shaven-beard" appearance. The liver exhibits areas of coagulative necrosis of considerable size; the adrenals and the kidneys are congested. The pleural cavity frequently contains an excess of clear fluid, and the lungs are mottled. The pericardial vessels are also injected and the sac contains an increased quantity of clear fluid. The distribution of the bacilli varies. With moderately virulent cultures they occur only in the local exudates in peritoneal and pleural cavities. In rare instances, indeed, they may disappear even from the abdominal cavity, be greatly reduced in numbers, absent from the internal organs and blood, or occur there in very small numbers. This disappearance may have taken place when death has occurred as early as twenty-four hours after inoculation. Larger doses or intensified cultures give rise to a moderate invasion of the



blood and organs. If the autopsy on these animals is delayed, especially in warm weather, an increase of bacilli in the blood rapidly takes place, so that erroneous results may be obtained. Within the local exudates the bacilli are surrounded by capsules and are often contained within polymorphonuclear leucocytes. The bacilli can also be cultivated from the fluid portions of the intestinal contents. The ingestion of cultures gives rise to no results unless the stomach-contents are first neutralized; in the latter case death may occur; the small intestine is hyperæmic; the contents are hemorrhagic and mucoid and the bacilli can be cultivated from them.

The *rabbit* usually responds with a localized swelling at the site of the subcutaneous injection, from which the animal usually recovers. When the injection results fatally the local infiltration resembles that in the guinea-pig, being, however, more marked than in that animal.

*Cats* also succumb to subcutaneous injections. Feeding alone produces no result. If, however, croton oil be first administered and the culture be then introduced into the stomach, diarrhoea sets in, the bacillus is recoverable from the dejections and death may result. In the last case the mucosa of the large intestine is hyperæmic and secretes an excess of mucus. The *dog* may succumb to simple feeding of the cultures. In positive instances diarrhoea sets in, the appetite is lost and death may take place in five or six days. The mucous membrane of the intestine is hyperæmic; hemorrhages occur, and the cavity of the gut contains a great excess of mucus from which the bacillus may be recovered.

The dead cultures are also toxic. Certain results of the inoculations into guinea-pigs suggest that the fatal effects are due to a toxic agent rather than to an infection *per se*. Cultures killed by exposure to temperatures of 60° C. for from fifteen to twenty minutes are still active. In the



course of certain immunization experiments one of the goats of a series succumbed to inoculation with dead cultures. On November 22d, 20 cc. of a bouillon culture, killed by heating to 60° C., were given under the skin of the shoulder. Considerable induration developed at the site of inoculation; diarrhœa set in, from which the animal seemed to recover. On November 29th, a second injection of 22 cc. of the culture was given; on the next morning the animal was dead. The *autopsy* showed edema over the site of inoculation. The nates were covered with thin, partly dried fecal matter. The mucous membrane of the gut was hyperæmic and presented numerous punctiform hemorrhages.

Dead cultures injected into rabbits and guinea-pigs cause: (1) elevation of temperature; (2) symptoms of intoxication (especially in guinea-pigs) which may come on within two of three hours after the injection; and (3), in rabbits, rapid recovery with a localized and decreasing swelling; in guinea-pigs, similar phenomena or death in a few hours or after four to six weeks. In the last instance the animals show great emaciation. In the case of those that have recovered from the immediate results of the injection agglutinating properties for the bacilli appear in the blood.

*Has this Bacillus been Found in other Epidemics of Dysentery?*—If the bacillus described is of significance in the etiology of dysentery it must occur with regularity in the disease. Whether or not it will be found to have the distribution that is necessary in order to establish this relationship can only be determined from studies carried on in widely different places and in all forms of the disease. That the bacillus is identical with the organism obtained by Shiga in the epidemic of dysentery which prevailed in Japan, there can be no reasonable doubt. In morphological, cultural, and pathogenic characteristics the two organisms are indistinguishable.

Through the courtesy of Dr. J. H. Musser I have been enabled to study, bacteriologically and pathologically, a case of chronic dysentery contracted during the Spanish war in Porto Rico. The patient, a soldier, entered the hospital of the University of Pennsylvania in December, 1899. His dysentery dated back some months; the movements were frequently examined for amœbæ with negative results. A brief abstract of the autopsy protocol is as follows:

The body is that of a greatly emaciated man, about thirty-five years old. *Dorsal decubitus*. The peritoneal cavity contains a small amount of reddish fluid. The colon is thickened, and in the peritoneal surface, especially along the sigmoid flexure, shows dark points and lines of discoloration. The rectum and sigmoid flexure are contracted; the tranverse colon, on the other hand, is dilated. The mucosa of the large gut is thickened throughout; in addition there are small, recent hemorrhages into its substance. There is no pronounced ulceration; the mucous membrane presents a granular aspect; there are superficial areas denuded of epithelium, and others, which are slate-colored and show dark pigmentation. The submucosa is not especially thickened except in the lower part of the gut, where there is much contraction. The mucous membrane of the transverse colon is edematous; the cecum is less affected than the colon, while the small intestine has entirely escaped. No pseudo-membrane is present except upon a small portion of the lower part of the sigmoid flexure.

The bacteriological examination made from the contents of the hepatic and sigmoid flexures gave growths in which the two general types of bacilli already described were contained. The predominating form agreed with Type II (*B. coli communis* group); in addition, there were colonies of an organism which in morphological, cultural,



and pathogenic characters, and in the agglutination reaction, corresponded with the variety of bacilli represented by Type I. The histological appearances in this case differ from those in the acute disease and equally from those of the amœbic variety. The changes are found more particularly in the mucosa and submucosa and represent, it would appear, a later stage in the course of the acute disease. Before describing the other changes it should be mentioned that a striking feature in the case is the congestion in the mucosa, submucosa, and muscularis. Numerous large veins, distended with blood, occupy the field of the microscope. Whether these vessels are newly formed cannot be stated positively; but they certainly are many times larger than any pre-existing vessels normally met with in the same situations. In a few places the surface of the mucosa shows a necrosis of the hyaline or coagulative variety, there being no appearances of exudative fibrin in these areas. These necroses do not include the entire thickness of the mucous membrane, but cap superficial foci. There can be no doubt that this tissue-death indicates an exacerbation of the acute disease, with which, indeed, the great congestion may be partially associated.

The chief and, as I take it, characteristic changes in this stage of the disease are proliferative in character. The mucous membrane is not markedly altered in volume. Its structure is, however, greatly modified. Very few glandular crypts remain. The membrane is represented by a mass of spindle and epithelioid cells together with a reticular and coarser intercellular network, enclosing the remains of the crypts of Lieberkühn. The submucosa, also, shows a new growth of tissue, in which, however, appear much more advanced changes. The submucosa is composed of dense, almost hyaline, and structureless tissue, taking a vivid eosin stain and enclosing foci of epithelioid cells. The hardening and distortion of the gut



were, doubtless, caused by this new growth of tissue and its subsequent contraction. The dilated blood-vessels, mentioned above, occupy a prominent place in this coat. A variable number of lymphoid, plasma, and eosinophilic cells occur, especially about the veins.

The muscular coat is also the seat of a multiplication of connective-tissue cells, which is shown by the masses of epithelioid cells separated by muscle-fibres, as well as an increase in foci of the fibrous tissue.

Blood-pigment is present both in the muscular and the subperitoneal coat.

The careful bacteriological studies in Egyptian dysentery made by Kruse and Pasquale contain numerous references to typhoid-like bacteria. Critical examination shows the majority to belong to the groups of *B. coli communis*. The typhoid characteristics depend largely on cultural resemblances—most marked in growths on agar-agar. Fermentation and their effects upon milk eliminate the suspicion that they may be typhoid bacilli, or the organism obtained by Shiga in Tokio, or by myself in Manila. Still other examples of bacilli, similar to and possibly identical with *B. dysenteriae* (Shiga), have been found in dysentery, though they are not suspected of standing in any etiological relation to it. Pansini studied four cases of abscess of the liver, three of which followed dysentery. The bacilli, which were isolated, resembled *B. typhosus*—indeed, Pansini could not distinguish between the two series. Babes also, although only in a single instance, isolated such an organism from a case of dysentery.

Since the publication of Shiga's studies, Escherich and Celli have both attempted to show that the organisms obtained from their respective epidemics of dysentery are identical with the *B. dysenteriae*. In both cases they have proceeded upon the false assumption that Shiga's

micro-organism was a variety of *B. coli communis*, whereas, in point of fact, it is much more nearly related in its cultural and physiological properties to *B. typhosus*.

The question naturally arises, In what way does it differ from *B. typhosus*? Comparison of the Eberth-Gaffky and Shiga bacilli show the criteria of difference to be by no means numerous. The main features, however, are as follows: The latter shows less marked motility when first isolated and a tendency to lose motility rapidly in artificial cultivations; it displays a more uniform generation of indol; after a brief preliminary acid production in milk it gives rise to a gradually increasing alkalinization; it is inactive to blood-serum from typhoid cases; but reacts with serum from dysenteric cases to which *B. typhosus* does not respond.

*The Agglutination Test.*—While the absolute value of this test in determining the specificity of bacteria may be open to doubt, its use in differentiation is now unquestioned. Undoubtedly there are limits to its usefulness, and experience (gained especially in typhoid fever) has shown that the changes upon which the property of the blood-serum depends for its evolution in certain instances may fail to take place. The tests in the case of the bacillus isolated in Manila were made at the time with blood obtained from acute and chronic cases of dysentery, occurring there and in the surrounding country. For carrying out the tests the blood was obtained in capillary tubes from the lobe of the ear of the living, and in larger quantities directly from the cavities of the heart by means of sterilized pipettes from the fatal cases of dysentery. The tests were made under the microscope and by growing the organisms in mixtures of bouillon and blood-serum. After our return to this country, the blood-serum from the case of Porto-Rican dysentery was employed and gave positive results. Through the



courtesy of Assistant-Surgeon Craig, stationed at the Presidio at San Francisco, I obtained capillary tubes filled with blood taken from convalescents and other soldiers suffering from chronic dysentery acquired in the Philippines. The present status of the agglutination reaction may be summed up as follows:

Positive results were obtained with cases definitely known to have been infected with the micro-organism in question. The results obtained from the blood derived from chronic dysentery were more variable. Dr. Osler has written me of his experience. In several cases of amoebic dysentery which have come under his charge in the Johns Hopkins Hospital, the blood-serum failed to produce the reaction with the bacillus obtained in Manila; in one case of the Porto-Rican disease a positive reaction was given.

The above results tend to emphasize the distinction of types of dysentery occurring in the tropics. They further tend to confirm the possibility that the acute dysenteries are caused by *B. dysenteriae*. To what extent the organism is concerned with the production of chronic dysentery remains to be established. That we must recognize a chronic form of tropical dysentery that is not in its entire course associated with the presence of amoebæ in large numbers, and that possesses totally different pathological lesions, is certain. I am inclined to the opinion that this type is not the commonest form of chronic tropical dysentery, and that it is less frequent than the amoebic type. As it appears to be the form that gives a positive serum reaction with *B. dysenteriae*, its extent and distribution may now be open to investigation.

Bearing directly upon these considerations are the results of Lieutenant Strong's studies continued after our departure from Manila. He writes: "After you left we



had a large number of acute cases of dysentery. It seems certain that this form, which we have begun to speak of as *acute infectious dysentery*, is independent of amœbæ. I have now records of fourteen cases (not all were fatal) which I studied bacteriologically. From the stools in all of these, there has been obtained a bacillus which agrees with the organism obtained by you. I have also obtained the organisms from the mesenteric glands in three fatal cases. In one case of acute dysentery, with secondary acute fibrinous peritonitis, I obtained it from the exudate. The agglutination reaction is not invariable. Amœbæ were never demonstrable in any of these fourteen cases. On the other hand, in every case with certain anatomical lesions we always find the amœbæ. In some cases of dysentery in which the amœbæ were absent and the bacilli present, that have lasted four to five weeks (one case lasted nearly two months) and then resulted fatally, we see a continuation of the same process that is observed in the acute fatal cases. The lesions are those of necroses of the mucous membrane and induration of the gut."

*Protective Inoculation and Serum Therapy.*—It is not unreasonable to hope that with the discovery of the specific cause of dysentery, particularly if it proves to be a bacterium capable of being artificially cultivated, means will be found by which protective inoculation may be carried out with effect and safety. The fundamental conditions underlying such immunization are now fairly established, and two general methods of accomplishing such results are open to investigation. In the first place, an active immunization may be achieved through the use of cultures of a determined grade of activity; in the second the serum of animals may be employed either as a therapeutic agent or to provide a passive immunity.

It has been found possible, through the use of cultures

destroyed by heat or the addition of chemicals (tricrosol), to protect small animals from subsequent inoculations with virulent bacilli. Larger animals, such as the goat, when treated first with the dead and afterwards with the living cultures, develop a gradually increasing resistance to the inoculations; their blood-serum assumes highly agglutinating qualities for the bacillus, and coincidentally acquires protective and healing properties. My own experiments relating to this topic have been carried out on small animals only. Shiga, has, however, been able to test the serum upon human cases. Dr. Eldridge<sup>1</sup> in his report gives the following figures: Up to November 1, 1899, Shiga had treated with the serums in 1898 in Laboratory Hospital, 65 cases, death-rate 9 per cent.; in 1899 in Laboratory Hospital, 91 cases, death-rate 8 per cent.; in 1899 in Hirowo Hospital, 110 cases, death-rate 12 per cent.; During the same period of 1899 there were under ordinary treatment at Tokio: At Honjo Hospital, 166 cases, death-rate 37.9 per cent.; at Hirowo Hospital, 53 cases, death-rate 37.7 per cent.; at Komogome Hospital, 398 cases, death-rate 34.6 per cent.; in private houses, 1,119 cases, death-rate 28.5 per cent.

I should, however, expect greater benefit from a species of vaccination, especially to those exposed to the endemic or endemo-epidemic dysentery of the tropics. The encouraging results of the injections of the dead bacilli of Asiatic cholera and typhoid fever render justifiable the use of a similar procedure in persons exposed to dysentery. The practical details of such inoculations will, of course, be established only after trials; preferably upon human beings who are anxious to submit to this method of treatment. I have found it possible to prepare cultures which after being killed possess a definite degree of toxicity for guinea-pigs. The only example of an experiment upon a

<sup>1</sup> *Public Health Reports*, vol. xv., No. I.



man yet available is that performed by Shiga, who directed that about  $\frac{1}{12}$  of an agar-culture, suspended in bouillon and killed by heat, should be injected into the subcutaneous tissues of his back. The immediate results of the injection were pain in the head, slight chill and fever, and local infiltration. After five or six days—the symptoms having in the interim entirely disappeared, except for some slight swelling—this area of infiltration increased and called for incision. The subcutaneous tissues were found thickened, indurated, and infiltrated with pus, which was sterile to cultures. The local lesion, similar to those in animals, was, it is thought, produced by the toxic substance contained within the dead bodies of the bacteria. Immediately after incision, all disagreeable symptoms subsided except the local infiltration, which disappeared gradually.

If this experiment can be taken as an index, the poison of *B. dysenteriae* is more active than the analogous substance contained in the bodies of the typhoid and Asiatic cholera organisms. This objection, if true, could be eliminated by dosage, or, if necessary, by combining the vaccine with immune-serum, as has recently been recommended by the German Plague commission in carrying out the inoculations with the Plague bacilli. Shiga's blood-serum, ten days after the injection, showed active agglutination of the bacilli.

An interesting, if somewhat disagreeable incident, was experienced by one of the laboratory assistants in Baltimore. In studying the acid production of the Manila bacillus a small quantity of fluid culture was aspirated into the mouth. The culture was expectorated and the mouth rinsed with a weak carbolic-acid solution. Notwithstanding this precaution, a severe diarrhoea, with bloody and mucous stools, pain and tenesmus developed within forty-eight hours. I was in Philadelphia at the



time, and the scientific ardor of the patient was so greatly depressed as a result of his discomfort and suffering that cultures were not made from the dejections, nor was I notified of the accident until several weeks afterwards.

Very little remains for me to say at this time. It is only natural to ask whether the foregoing considerations justify a belief in a specific organism of dysentery. My own sense is against that belief, although it must be conceded that the varieties of the diseases are fewer than the clinical and pathological-anatomical conceptions of the time would lead one to suppose. Excluding the sporadic cases, which need a much closer bacteriological study than has yet been accorded to them, it is entirely possible that two specific organisms may be responsible for the epidemic and endemic diseases *per se*. I think that I have shown that tropical dysentery consists of a bacillary and an amoebic form, separable in their early and their later stages by their clinical histories, their etiology and pathological anatomy. It is important to know whether the epidemic disease is more uniform in its causation and pathological anatomy. The studies of the Japanese disease by Shiga are highly suggestive of this interpretation, but additional observations will be required before we can accept as final his conclusions.



# INDEX.

	PAGE
<b>Abortion</b> , tubal (Boldt) .....	294
<b>Abscess</b> , cerebellar (Biggs) .....	287
“ hepatic (Le Wald) .....	24
<b>Acid Intoxication</b> (Herter and Wakeman) .....	226
<b>Actinomycosis</b> , case of (Mandelbaum) .....	178
<b>Addison's Disease</b> , case of (Le Wald) .....	237
“ “ with atrophy of adrenal (Phillips) .....	5
<b>Adenoma</b> of thyroid (Le Wald) .....	24
<b>ADLER</b> . Primary growths of pleura and lung .....	283
<b>Adrenal</b> , aberrant (Wood) .....	170
“ atrophy of, in Addison's disease (Phillips) .....	5
“ deposits in liver (Noyes) .....	4
<b>Albumin</b> , precipitation of, by bacteria (Libman) .....	241
<b>Amyloid Degeneration</b> , chemistry of (Levene) .....	III
<b>Aneurism</b> , aortic (Carter) .....	233
“ “ (Janeway) .....	273
“ “ (Lambert) .....	134
“ dissecting of aorta .....	3
“ “ with rupture into pulmonary artery (Conner) .....	54
<b>Anthracosis</b> of lung (Hodenpyl) .....	79
<b>Antitoxins</b> , relations of, to globulins (Atkinson) .....	73
<b>Antrum</b> (Highmore), carcinoma of (Le Wald) .....	99
<b>Aorta</b> , aneurism of (Carter) .....	233
“ “ “ (Janeway) .....	273
“ “ “ (Lambert) .....	134
“ rupture of aneurism of (Le Wald) .....	2, 3
<b>Appendix Vermiformis</b> , foreign body in (Larkin) .....	106
“ “ stenosis of (Möschowitz) .....	1
“ “ tuberculosis of (Hodenpyl) .....	182
<b>Arterio-sclerosis</b> , case of (McAlpin) .....	192
<b>Arthritis Deformans</b> , case of (Thayer) .....	275
<b>Asexualism</b> , case of (Brooks) .....	53
<b>Asphyxiation</b> from tobacco plug in bronchus (Le Wald) .....	280
<b>ATKINSON</b> . Antitoxic globulins .....	73
<b>Atresia</b> of duodenum (Thacher) .....	101



	PAGE
<b>Atrophy</b> of adrenal (Phillips).....	5
“ “ brain (Bovaird).....	31
<b>Bacillus Aërogenes Capsulatus</b> in puerperal infection (Wood).....	25
“ <b>Pestis</b> , cultures of (Park).....	108
“ <b>Tuberculosis</b> , growth of, on Hess's medium (Wilson)....	123
“ “ nucleic acid in (Levene).....	240
“ <b>Typhosus</b> in uterus (Lartigau).....	137
“ “ persistence in ice (Park).....	195
<b>BAILEY.</b> Sarcoma of thigh.....	271
“ Uterine nodule.....	194
<b>BATES.</b> Adrenal extract as hæmostatic.....	278
<b>BIGGS.</b> Iliac thrombus.....	57
“ Carcinoma of liver.....	105
“ Cerebellar abscess.....	287
“ Suppurative metritis.....	136
“ Tuberculosis of bile ducts.....	263
“ Tuberculous endometritis.....	259
“ Tuberculous spleen.....	288
<b>Bile Ducts</b> , tuberculosis of (Biggs).....	263
<b>Bladder</b> , diverticulum of (Moschowitz).....	71
<b>Blood</b> , collection of, for examination (Thacher).....	276
“ effect of, in animals without adrenals (Levin).....	241
<b>BOLDT.</b> Gangrenous dermoid.....	136
“ Intestinal intussusception.....	135
“ Tubal abortion.....	294
<b>Bone</b> , sarcoma of (Sayre).....	61
<b>BOVAIRD.</b> Cystic kidney.....	250
“ Hemiatrophy of brain.....	31
“ Intubation.....	249
“ Intestinal tuberculosis.....	123
<b>Breast</b> , gelatinous carcinoma of (Hodenpyl).....	62
“ osteocarcinoma of (Larkin).....	176
<b>Bronchitis</b> , streptothrix (Norris and Larkin).....	15
<b>Bronchus</b> , cast of (Janeway).....	232
<b>BROOKS.</b> Case of asexualism.....	53
“ Case of trichinosis.....	167
“ Case of Weil's disease.....	10
“ Death from ether.....	261
“ Duodenal ulcer.....	81
“ Fibromatosis.....	22
“ Malposition of kidney.....	113
“ Pin in appendix.....	57
<b>BUXTON.</b> Photographs of filaria.....	251
<b>Carcinoma</b> of antrum of Highmore (Le Wald).....	99

	PAGE
<b>Carcinoma</b> (gelatinous) of breast (Hodenpyl) .....	62
“ (osteo) of breast (Larkin) .....	176
“ of liver (Biggs) .....	125
“ (primary) of lung (Hodenpyl) .....	270
“ (gelatinous) of lung (Larkin) .....	66
“ (primary) of pancreas (Hodenpyl) .....	182
“ (gelatinous) of peritoneum (Larkin) .....	66
“ of prostate (Conner) .....	266
“ of stomach (Larkin) .....	19, 66
<b>CARTER.</b> Aortic aneurism .....	233
<b>Cast</b> , bronchial (Janeway) .....	232
<b>Celloidin</b> , new method of embedding in (Hodenpyl) .....	277
<b>Cerebellum</b> , abscess of (Biggs) .....	287
<b>Cirrhosis</b> of liver (Wood) .....	60, 245
<b>Coccytis</b> , tuberculous (Jeffries) .....	130
<b>Cold</b> , action on bacteria (Park) .....	273
<b>Colloid</b> , chemistry of (Levene) .....	111
<b>Committee</b> (microscopy) report on case of Mr. Kelly .....	52
<b>CONNER.</b> Aortic aneurism (rupture of) .....	54
“ Cysts of kidney .....	160
“ Double spleen .....	102
“ Duodenal ulcer .....	102, 265
“ Endocarditis (malignant) .....	61
“ Liver cysts .....	160
<b>Coronary Artery</b> , occlusion of (Wood) .....	33
<b>Cyst</b> of the kidney (Bovaird) .....	250
“ “ “ “ (Conner) .....	160
“ “ “ stomach (Schultze) .....	260
<b>Degeneration</b> , fatty, of heart (Dunham) .....	28
<b>Dermoid</b> , gangrenous (Boldt) .....	136
<b>Diabetes</b> , coma in (Herter) .....	267
<b>Diastasis</b> of caput femoris (Gibney) .....	294
<b>Diphtheria Bacillus</b> , morphology of (Park) .....	73
<b>Diverticulum</b> of bladder (Moschowitz) .....	71
“ “ intestine (Hodenpyl) .....	182
<b>DUNHAM.</b> Tuberculosis of heart .....	169
“ Fatty heart .....	281
<b>Duodenum</b> , atresia of (Thacher) .....	101
“ ulcer of (Conner) .....	102, 265
“ “ “ (Brooks) .....	8
“ “ “ (Lartigau) .....	164
<b>Dysentery</b> , Etiology of (Flexner) .....	297
<b>Elephantiasis</b> of ears (Hodenpyl) .....	94

	PAGE
<b>Embolism</b> (fat) of pulmonary artery (Larkin) . . . . .	159
<b>Encephalitis</b> , case of (Hodenpyl) . . . . .	32
<b>Endocarditis</b> , malignant (Conner) . . . . .	61
<b>Endometritis</b> , tuberculous (Biggs) . . . . .	259
<b>Endothelioma</b> , remarks upon (Wood) . . . . .	270
<b>Epiglottis</b> , tuberculosis of (Ewing) . . . . .	97
<b>Epithelioma</b> of heart (Wood) . . . . .	246
"    "    "    oesophagus (Hodenpyl) . . . . .	175
<b>Ether</b> , death from (Brooks) . . . . .	261
<b>EWING.</b> Papilloma of larynx . . . . .	94
"    Thrombosis superior mesenteric artery . . . . .	97
"    Tuberculosis of epiglottis . . . . .	97
<b>Fibromata</b> , multiple (Brooks) . . . . .	22
<b>Filaria Sanguinis</b> , photos. of (Buxton) . . . . .	251
<b>Filariasis</b> , case of (Nicoll) . . . . .	169
<b>FLEXNER.</b> Etiology of dysentery . . . . .	297
<b>FRANK.</b> Neuron retraction . . . . .	89
<b>FREEBORN.</b> Staining technique . . . . .	170
<b>Gangrene</b> of lung (Le Wald) . . . . .	279
<b>Genitalia</b> , tuberculosis of (Moschowitz) . . . . .	72
<b>GIBNEY.</b> Diastasis caput femoris . . . . .	294
"    Osteomyelitis femoris . . . . .	294
<b>Globulins</b> , antitoxic (Atkinson) . . . . .	73
<b>Heart</b> , congenital lesion (Potter) . . . . .	254
"    fatty (Dunham) . . . . .	281
"    rupture of (Potter) . . . . .	234
"    tuberculosis of (Dunham) . . . . .	169
<b>Hemiatrophy</b> of brain (Bovaird) . . . . .	31
<b>Hemorrhage</b> into adrenals (Norris) . . . . .	179
<b>Hermaphrodite</b> (pseudo), (Mathews) . . . . .	42
<b>HERTER.</b> Acid intoxication . . . . .	226
"    Diabetic coma . . . . .	267
<b>Hesse's Medium</b> , growth of tubercle bacillus in (Wilson) . . . . .	123
<b>HODENPYL.</b> Anthracosis . . . . .	79
"    Carcinoma (gelatinous) of breast . . . . .	62
"    (primary) of lung . . . . .	270
"    of pancreas . . . . .	182
"    Clove-oil celloidin embedding . . . . .	277
"    Epithelioma oesophagus . . . . .	175
"    Meningo encephalitis . . . . .	32
"    Oesophageal varices . . . . .	180
"    Pneumothorax . . . . .	181
"    Tuberculosis of appendix . . . . .	182
"    "    "    pleura . . . . .	42



	PAGE.
<b>Iliac Artery</b> , thrombosis of (Biggs).....	57
<b>Infarction</b> of myocardium (Wood).....	33
“ “ small bowel (Le Wald).....	75
<b>Infection</b> , puerperal (Wood).....	25
“ typhoid, of uterus (Lartigau).....	137
<b>Intestine</b> , diverticula of (Hodenpyl).....	182
“ hyperplastic tuberculosis of (Lartigau).....	183
“ intussusception (Boldt).....	135
“ sarcoma of (Libman).....	197
“ tuberculosis of (Bovaird).....	123
<b>Intoxication</b> in diabetes (Herter).....	226
<b>Intubation</b> , effects of (Bovaird).....	249
<b>Intussusception</b> , intestinal (Boldt).....	135
<b>JANEWAY.</b> Aortic aneurism.....	273
“ Bronchial cast.....	232
“ Lymphatic leukæmia.....	115
<b>JEFFRIES.</b> Malaria from wound infection.....	129
“ Tuberculous coccytis.....	130
<b>Kidney</b> , absence of (Le Wald).....	70
“ “ “ (Schultze).....	282
“ cystic (Bovaird).....	250
“ “ (Conner).....	160
“ horse-shoe shaped (Thayer).....	274
“ lympho-sarcoma (Wood).....	243
“ malposition of (Brooks).....	113
<b>Knee</b> , osteosarcoma of (Sayre).....	61
<b>LAMBERT.</b> Aneurism of aorta.....	134
“ Laryngitis in typhoid.....	132
<b>LANGMANN.</b> Papilloma of ovary.....	238
<b>LARKIN.</b> Carcinoma (gelatinous) of lung.....	66
“ “ “ “ peritoneum.....	66
“ “ “ “ stomach.....	66
“ “ “ “ “.....	19
“ Embolism of pulmonary artery.....	159
“ Foreign body in appendix.....	106
“ Hemorrhage of myocardium.....	36
“ Myocarditis.....	38
“ Osteocarcinoma of breast.....	176
“ Pancreatitis.....	19, 20, 21
“ Thrombosis of pulmonary artery.....	158
<b>LARTIGAU.</b> Duodenal ulcer.....	164
“ Hyperplastic tuberculosis of intestine.....	183
“ Typhoid infection of uterus.....	137

	PAGE
<b>Laryngitis</b> in typhoid (Lambert).....	132
<b>Larynx</b> , foreign body in (Le Wald).....	53
"    papilloma of (Ewing).....	94
<b>Leukæmia</b> , case of (Janeway).....	115
LEVENE. Chemistry of mucoid, colloid, etc.....	111
"    Nucleic acid in tubercle bacillus.....	240
LEVIN. Adrenals in relation to blood toxicity.....	24
"    Mucinæmia.....	109
LE WALD. Abscess of liver.....	24
"    Adenoma of thyroid.....	24
"    Carcinoma antrum of Highmore.....	97
"    Double uterus.....	70
"    Four cusps of pulmonary valve.....	3
"    Gangrene lung.....	279
"    Infarction of small intestine.....	75
"    Rupture of aorta.....	2, 3
"    Skin sarcoma.....	40
"    Transposition of viscera.....	24
"    Tuberculous peritonitis.....	235
LIBMAN. Precipitation of albumin by bacteria.....	241
"    Pulmonary stenosis.....	247
"    Sarcoma of small intestine.....	197
<b>Liver</b> , abscess of.....	24
"    adrenal deposits in (Noyes).....	4
"    carcinoma of (Biggs).....	105
"    cirrhosis of (Wood).....	60, 245
"    cystic (Conner).....	160
<b>Lung</b> anthracosis (Hodenpyl).....	79
"    gelatinous carcinoma of (Larkin).....	66
"    primary carcinoma of (Hodenpyl).....	270
"    "    neoplasms of (Adler).....	283
<b>Lymph Nodes</b> , perforation from, into trachea (Mathews).....	131
<b>Lympho-sarcoma</b> of kidney (Wood).....	243
<b>Malaria</b> , relation to wound infection (Jeffries).....	129
MANDELBAUM. Case of actinomycosis.....	178
MATHEWS. Bronchial gland tuberculosis.....	131
"    Pseudo-hermaphrodite.....	42
"    Tumor of nymphæ.....	70
McALPIN. Case of arterio-sclerosis.....	192
"    Perforation of stomach by toothpick.....	193
<b>Meningitis</b> , pneumococcus (Wood).....	59
"    with encephalitis (Hodenpyl).....	32
<b>Mesenteric artery</b> , thrombosis of (Ewing).....	94
<b>Metritis</b> , suppurative (Biggs).....	136

	PAGE
Middleton-Goldsmith Lecture (Flexner).....	297
Milk, formalin as preservative of (Park).....	252
Mole, uterine (Bailey).....	194
MOSCHOWITZ. Diverticulum of bladder.....	71
“ Stenosis of appendix.....	1
“ Tuberculosis of genital tract.....	72
Mucinæmia (Levin).....	109
Mucoid substances, chemistry of (Levene).....	111
Myocarditis, interstitial (Larkin).....	36, 38
“ tuberculous (Wood).....	54
Myocardium, infarction of (Wood).....	33
Myomalacia (Phillips).....	34
Necrosis (fat) of pancreas (Larkin).....	19
Neuron, death of (Van Gieson).....	156
Neuron retraction (Frank and Weil).....	89
“ “ (Van Gieson).....	85
NORRIS. Adrenal hemorrhages.....	179
“ Streptothrix bronchitis.....	15
NORTHRUP. Bone in œsophagus.....	59
NOYES. Adrenal deposits in liver.....	4
Nucleic acid in tubercle bacilli (Levene).....	240
Œsophageal varices (Hodenpyl).....	180
Œsophagus, epithelioma of (Hodenpyl).....	175
Œsteomyelitis of femur (Gibney).....	294
Ovary, papilloma of (Langmann).....	238
Pancreas, carcinoma of (Hodenpyl).....	182
Pancreatitis, hemorrhagic (Larkin).....	19
“ interstitial (Thayer).....	274
“ suppurative (Larkin).....	20, 21
Papilloma of larynx (Ewing).....	94
“ of ovary (Langmann).....	238
Paraffin in bacteriology technique (Park).....	293
PARK. Action of cold on bacteria.....	293
“ Formalin as milk preservative.....	252
“ Morphology of diphtheria bacillus.....	73
“ Plague bacillus cultures.....	106
“ Typhoid bacillus in ice.....	195
Peritoneum, gelatinous carcinoma of (Larkin).....	66
“ sarcoma of (Wood).....	243
Peritonitis, tuberculous (Le Wald).....	235
PHILLIPS. Addison's disease.....	5
“ Myomylacia.....	34
Pleura, neoplasms of (Adler).....	283
“ tuberculosis of (Hodenpyl).....	43



	PAGE
<b>Pneumothorax</b> , case of (Hodenpyl).....	181
POTTER. Congenital lesion of heart.....	254
"    Ruptured heart.....	234
"    "    spleen.....	255
<b>Prostate Gland</b> , carcinoma of (Conner).....	266
<b>Pulmonary artery</b> , fat embolism of (Larkin).....	159
"    "    thrombosis of (Larkin).....	158
<b>Pulmonary stenosis</b> , case of (Libman).....	247
<b>Pulmonary valve</b> with four cusps (Le Wald).....	3, 24
<b>Sarcoma</b> of knee (Sayre).....	61
"    "    peritoneum (Wood).....	243
"    "    skin (Le Wald).....	40
"    "    small intestine (Libman).....	197
"    "    thigh (Bailey).....	271
SCHULTZE. Absence of kidney.....	282
"    Cyst of stomach.....	260
<b>Skin</b> , sarcoma of (Hodenpyl).....	41
"    "    "    (Le Wald).....	40
<b>Spleen</b> , double (Conner).....	102
"    rupture of (Potter).....	255
"    tuberculosis of (Biggs).....	288
<b>Stains</b> , preparation of (Freeborn).....	170
<b>Stomach</b> , gelatinous carcinoma of (Larkin).....	66
"    cyst of (Schultze).....	260
"    perforation of (McAlpin).....	193
<b>Streptococcus</b> , new variety of (Libman).....	196
<b>Streptothrix</b> , bronchitis (Norris and Larkin).....	15
<b>Subclavian artery</b> , thrombosis of (Le Wald).....	280
THACHER. Atresia of duodenum.....	101
"    Collection of blood for examination.....	276
"    Stricture of urethra.....	101
THAYER. Arthritis deformans.....	275
"    Horse-shoe shaped kidney.....	274
<b>Thrombosis</b> of iliac artery (Biggs).....	57
"    "    pulmonary artery (Larkin).....	158
"    "    subclavian artery (Le Wald).....	280
"    "    superior mesenteric artery (Ewing).....	97
<b>Thyroid gland</b> , adenoma of (Le Wald).....	24
<b>Transposition</b> , visceral (Le Wald).....	24
<b>Trichinosis</b> , cases of (Brooks).....	167
<b>Tuberculosis</b> of bile ducts (Biggs).....	263
"    "    endometrium (Biggs).....	259
"    "    epiglottis (Ewing).....	97
"    "    genital tract (Moschowitz).....	72

# INDEX.

347

	PAGE
<b>Tuberculosis</b> (hyperplastic) of intestine (Lartigau).....	133
“ of heart (Dunham).....	169
“ “ (Wood).....	54
“ “ intestine (Bovaird).....	125
“ “ peritoneum (Le Wald).....	235
“ “ pleura (Hodenpyl).....	43
“ “ spleen (Biggs).....	288
<b>Typhoid Fever</b> , infection of uterus in (Lartigau).....	137
“ “ laryngitis (Lambert).....	132
<b>Ulcer</b> of duodenum (Brooks).....	81
“ “ “ (Conner).....	102, 265
“ “ “ (Lartigau).....	164
“ “ “ (Larkin).....	164
<b>Urethra</b> , stricture of (Thacher).....	101
<b>Urine</b> , chylous (Nicoll).....	169
“ infection of (Williams).....	289
<b>Uterus</b> , double (Le Wald).....	70
“ gas bacillus, infection of (Wood).....	25
“ suppurative, inflammation of (Biggs).....	136
“ typhoid, infection of (Lartigau).....	137
<b>Vaccine bodies</b> (Williams).....	165
<b>VAN GIESON</b> . Death of neuron.....	156
“ Neuron retraction.....	85
<b>Weil's disease</b> , case of (Brooks).....	10
<b>WILLIAMS</b> . Infection of urine.....	289
“ Vaccine bodies.....	165
<b>WILSON</b> . Tubercle bacillus on Hesse's medium.....	123
<b>WOOD</b> . Aberrant adrenal.....	170
“ Accessory lobe of liver.....	245
“ Carcinoma of heart.....	60, 245
“ Coronary artery disease.....	33
“ Tuberculosis of myocardium.....	54
“ Pneumococcus meningitis.....	59
“ Puerperal infection (gas bacillus).....	25
“ Remarks on endothelioma.....	270
“ Sarcoma of kidney.....	243
“ “ “ peritoneum.....	243

CANCELLED

THE  
LIBRARY OF THE  
MUSEUM OF  
COMPARATIVE ZOOLOGY  
AT HARVARD UNIVERSITY  
CAMBRIDGE, MASS.













RB  
1  
N32  
1899-  
1900

New York Pathological  
Society  
Proceedings

Biological  
& Medical  
Serials

PLEASE DO NOT REMOVE  
CARDS OR SLIPS FROM THIS POCKET

---

UNIVERSITY OF TORONTO LIBRARY

---



